Digitized by the Internet Archive
in 2018 with funding from
University of Toronto

https://archive.org/details/medjournalJan1926uoft

The University of Toronto Medical Journal

VOL. III JANUARY, 1926 NO. 3

EDITORIAL

SPECIFIC CHARACTER OF MALIGNANT NEOPLASIA*

AMONG the many investigators who for years have sought some one factor as responsible for malignant neoplastic growth there are some who have long given up this contention. Believing that the development of malignant new growth was a "specific process" with many causative factors combined to effect its successful development, Mr. Blair Bell set out to discover the "nature of cancer". To do this he chose first to study the nature of the normal tissue cell, its embryological characteristics and its physiological function.

It was observed that the normal, healthy cell reacted in different ways to different stimuli. Excessive stimulation acting on normal, healthy cells produced either a hyperplasia or a benign new growth between which no difference could be made out, and whose function was still essentially normal. If the cell, which was functioning abnormally due to some pathological change, was subjected to excessive stimulation, the resulting development was productive of malignant neoplasia. That is, cancer arises only in relation to the impaired body cell in its struggle to survive after injury from metabolic or extrinsic causes. As an example of the metabolic causative factor, Mr. Blair Bell has referred us to the development of the human ovum.

The function of the chorionic epithelium is to find nutrition for the ovum and for itself. This it does until the source of food supply can be taken care of by the embryo itself. If after this process of struggle the growth of the primitive, undifferentiated, nutritive cells of the chorion is not stopped by whatever factor is responsible, they may at this point lose their identity and manifest malignant characteristics. So, every normal body cell forced to adapt itself to altered conditions of deprivation or injury, in its struggle for existence, may revert to this simple "nutritive-seeking" type observed in the chorion. Unlike the chorion, there is no inhibitory factor to prevent its taking on a "riotous behaviour" and rapidly becoming malignant. The greater the reversion of the neoplastic cell to the characteristics of the trophoblast, the more nearly does its virulence compare with the highly virulent chorion-epithelioma.

On a basis of the above hypothesis a comparative chemical analysis was made of normal tissue, innocent and malignant neoplasm and chorionic villi. The findings showed a high content of phosphatides in both malignant neoplasm and chorionic epithelium. The direct application of this finding was made by Gye and Cramer, who showed that the rate of growth of malignant tumour in the mouse was directly proportional to the phosphatide content of the tumour.

A fact of greater significance, one on which the therapeutic aspect of cancer has a direct bearing was revealed from the study of the factors which determine the permeability of the cell membrane. It was found that whenever there was a high phosphatide-cholesterol ratio the cell membrane showed a high permeability. Malignant cells and chorionic epithelium exhibited this property to a degree three times as great as normal tissue. Moreover, the water content of these cells was high, a factor which, together with high permeability of the cell membrane, was contributory to rapid growth. Chorionic epithelium then is physiologically super-malignant.

Having studied the growth process in normal and in malignant neoplastic cells, and having made a comparative analysis of the chemical content of each and its relation to the growth process, a direct application was awaiting.

For many years a high abortion rate has been observed among women workers in the lead industry. From this observation the author was led to suspect that lead had some direct destructive effect on chorionic epithelium. Knowing the close kinship of this tissue to the malignant neoplasm the question arose as to whether lead should not act in a similar way to destroy cancer.

After proving the specific inhibitive effects of lead on plant growth and frog embryo by exposure to weak solutions of the metal, and having noted the stunting produced in children who had been poisoned by lead, it remained to apply the test to the specific growth process exhibited by malignant neoplasia. Two cases of the inoperable variety are reported, the one a carcinoma of the breast in the stage of ulceration with wide glandular involvement, the other a spindle-celled sarcoma with diffuse abdominal spread. The former case, after intravenous lead injections, showed diminishing signs of disease and, since five years have elapsed, may be considered a cure. The second case has greatly improved, with no present symptoms, a decided decrease in the size of the tumour and a marked gain in weight. The report of many more cases so treated await publication.

There is then no doubt that the secret as to the specific character of malignant neoplasia has been discovered.

Unquestionably, it marks a step in the advance of medical science that is without precedent.

*Prepared from an address by Prof. W. Blair Bell, B.S., M.D. Lond., delivered before the Academy of Medicine at Toronto on Nov. 10th, 1925.

LIPIODOL IN X-RAY DIAGNOSIS OF BRONCHO-PULMONARY LESIONS

THE first endeavour to outline the bronchial tree by X-ray methods, was by Jackson in 1918, who used dry bismuth insufflation. In 1920, a bismuth mixture in oil, injected by way of the bronchoscopic syringe, was used by Lynch. Sicard and Forestici in 1922 used the substance lipiodol, by intrathecal injection, to localize tumors involving the spinal canal. Sergent and Cottenot in 1923 were first to use lipiodol in the diagnosis of lesions of the bronchial tree, injecting the substance intra-tracheally, through the cricothyroid membrane.

The substance lipiodol is not a solution nor an emulsion, but a chemical combination of iodine and poppy oil—containing 40% of iodine. Its value lies in its opacity to X-rays.

At the Radiological Department, Toronto General Hospital, lipiodol is being used in diagnosis of broncho-pulmonary lesions, by intratracheal injection, *via* the cricothyroid membrane. At other institutions injection by way of the bronchoscopic syringe is employed. Ten to thirty cubic centimetres of warmed lipiodol are injected into one or both lungs—the patient then being turned from side to side so that the oil will disperse evenly. Then, immediately following this, X-ray plates are taken in various positions. A spasm of coughing will disseminate the oil throughout the lung, causing it to be forced from the lower to the upper lobes.

Lipiodol is especially efficacious for this work, as there is very little if any pain complained of, following injection, and scarcely any after-effects. The lung tissues appear remarkably tolerant to this substance—the absence of irritation being shown by the fact that the normal individual after lipiodol injection is troubled very little by coughing. This is believed to be due to the very slow liberation of the iodine from the chemical poppy oil iodine compound. The iodine liberated in this way is considered beneficial to the tissues—and lipiodol injection is only contra-indicated in cases of hyperthyroidism. Some authorities believe that lipiodol has an effect in diminishing the quantity of exudation in bronchoectasis, although this fact has not definitely been proven. The value of the lipiodol method consists in the fact that fairly accurate X-ray diagnosis of broncho-pulmonary lesions may be made. There is a marked contrast between X-ray plates taken before, and those taken after lipiodol injection; and also between normal and diseased lung structures. The pathological changes in bronchiectasis may be seen at a glance—the dilated and sacculated bronchi showing up very clearly. In pulmonary tuberculosis, by use of lipiodol injection, it is now considered that the differentiation between intra-pulmonary cavities, and localized pneumothoraces may be made—thus aiding in solving the problem of annular shadows observed in X-ray plates of suspected tuberculous lungs. Some authorities believe that early localization of lung abscesses is possible by this method of lipiodol injection—wherever the abscess cavity communicates with a bronchial ramification of sufficient size to allow the passage of the lipiodol. The diagnosis of interlobar empyema, new growth, etc., is also aided by the shadows cast on the fairly conspicuous bronchial tree.

Dr. Richards of the Radiological Department of the Toronto General

Hospital further informs us that lipiodol is now being fairly extensively used in France to outline by X-ray methods many other body cavities. It is employed to outline the ventricles and subarachnoid spaces of the brain; intrathecally to ascertain presence of tumors, etc., of the spinal cord and vertebrae; in examining the cavity of the bladder, ureters and pelvis of the kidney; in outlining the male urethra and seminal vesicles, and in the female the cavity of the uterus and lumina of the Fallopian tubes, and so on. It is expected that lipiodol will be more extensively used in this country, as soon as it becomes more available; as at present it is quite expensive and difficult to obtain.

In conclusion we desire to acknowledge the use of certain facts obtained from two articles regarding the use of lipiodol in X-ray diagnosis of broncho-pulmonary lesions in the *Journal of the Canadian Medical Association.*

HAEMATURIA

ROBIN PEARSE, F.R.C.S.

Demonstrator in Clinical Surgery, University of Toronto.

IT cannot be too often repeated, that haematuria is a symptom and not a disease, the haematuria will cease spontaneously in the majority of cases, but the disease is too often an unrecognized renal or vesical tumor which is allowed to progress to a state where cure is impossible and even temporary relief uncertain. Very few patients fail to consult a physician on first seeing blood in the urine; we do justice neither to our patients nor ourselves unless we make every effort to arrive at a complete diagnosis on the earliest possible occasion.

The urinary tract is, for clinical purposes, conveniently divided into the upper tract comprising kidneys and ureters, and the lower tract consisting of bladder and urethra, the urethra is further divided by the compressor urethrae or "cut off" muscle into anterior urethra and posterior urethra, the bladder and posterior urethra being for practical purposes one cavity.

Haematuria may be "occult", that is, discernible only on microscopic examination of a sedimented specimen, or "ocular", that is, the blood is sufficient in quantity to discolour the urine and be visible to the naked eye, the colour may vary from a smoky urine to the colour of blood.

In a voided specimen an occult haematuria gives no clue to the site of the lesion, a little consideration will show that the reverse is true in ocular haematuria. When bleeding occurs in the kidney, the blood is extruded at the same time as the urine, is intimately mixed with the urine as it passes to the bladder, will remain so in the bladder, and when the patient voids the stream must of necessity be uniformly discoloured throughout the whole act of micturition; this is termed "intimate haematuria". If on the contrary, there be some lesion present, such as a papilloma, in the bladder, then when the urine is voided, the bladder will contract upon the growth and crush it, in its effort to void it, the growth will in consequence bleed, and the blood will be extruded with or after the last few drops of urine, producing a phenomenon conveniently called "terminal haematuria". Of course a bladder tumour or ulcer may at times break or ulcerate into a small blood vessel, giving a continuous stream of blood into the bladder which mixes with the urine as it collects there, and when the patient voids the stream will resemble that from a renal haematuria, but careful observation will usually show that after all the urine is passed the patient has considerable tenesmus and passes some pure blood, often accompanied by clots, from the emptied bladder.

The first step in examination should therefore be to have the patient void into two glasses, the bulk of the urine being passed into the first glass, this constitutes the two-glass test for blood. If the blood is terminal it will be confined to the last glass, and one considers the bladder lesions which might produce haematuria, acute prostatitis or posterior urethritis,

acute cystitis (rarely a primary condition), stone, new growth, and enlarged prostate. A history of frequency and difficulty in an elderly man suggests enlarged prostate, rectal examination and estimation of residual urine is indicated. Urgency and frequency by day with pain referred to the tip of the penis suggests stone in the bladder, and terminal haematuria with little or no disturbance suggests an early growth. In the former case cystoscopy is indicated, in the latter it is imperative.

It is worth noting that haematuria in a patient with an enlarged prostate occurs more frequently with the adenomatous enlargement than with the carcinomatous; there are three or four large vessels in the mucosa passing between the trigone and the posterior urethra; as the adenoma grows and projects into the cavity of the bladder it stretches these vessels and they give way when the patient strains to void, a carcinoma starts as a rule on the rectal surface of the gland and cannot produce haematuria until it has reached the vesical surface of the gland and ulcerated.

It is not advisable to cystoscope patients with enlarged prostates as a routine examination, both the local and general condition of the patient may contraindicate it, at the same time one must remember that a carcinoma of the bladder may accompany an adenoma, or arise as an extension of a carcinoma of the gland. Furthermore, when an extensive carcinoma is present in the bladder, cystoscopy is impractical, attempts at examination only result in straining and bleeding and nothing can be seen. In these cases a cystogram may help, the bladder is catheterised and filled with an emulsion of bismuth in liquid petrolatum, stereoscopic pictures are taken and the emulsion allowed to run out, the bladder is then filled with air and pictures again taken, filling defects from growths of moderate or large size or from prostatic enlargements are clearly seen, as also are irregularities in the bladder outline produced by diverticula.

Finally in a few cases exploratory cystotomy may be required to establish a diagnosis.

Turning now to a case of intimate haematuria, we have to consider lesions local to the kidney which produce haematuria, and the various systemic diseases of which haematuria may be an incident. Microscopic examination of the urine may show in addition to the blood, many pus cells and no pyogenic organisms, we should at once suspect a tuberculous kidney. There may be many sharp envelope-shaped crystals of calcium oxalate, and perhaps the patient complains of pain in the kidney or along the course of the ureter, or burning in the urethra on micturition, and enquiry as to diet may disclose a recent feast of strawberries. In any case the patient should be X-rayed for possible stone, the whole urinary tract on both sides should be taken, an oxalate stone throws the densest shadow, then phosphate and carbonate, while a pure uric acid or urate stone throws no shadow and can only be demonstrated by the X-ray as a filling defect in a pyelo-ureterogram. Fortunately pure urate stones are rare.

Apart from crystals, calculi and tuberculous lesions, it is remarkable how few renal haematurias show localizing symptoms, hence the term "symptomless haematuria". A growth in the kidney frequently forms

quite a large abdominal tumour before the patient discovers its presence.

In the absence of localising symptoms, and if palpation of the abdomen is negative, it is of great help to cystoscope the patient before the haematuria has stopped. The ureteral orifices can then be watched and the blood stained efflux seen to come from one or both ureters. Should it be unilateral, a pyelogram should be made to discover a possible early renal tumour too small to be palpable per abdomen, this would show as a distortion or obliteration of one or more calyces. Care must be taken to distinguish between obliteration of the cavity and mere lack of filling due to faulty technique.

Although of necessity considerable stress has been laid on the use of the cystoscope in the diagnosis of the source of haematuria, it must not be considered the first step in diagnosis, the rotation should be, as in every other process, from the simple to the more complex method. History taking, clinical examination, and complete examination of the urine should invariably precede cystoscopy; particular attention should be paid to any special tests which may be indicated during the progress of the clinical examination, such as blood pressure estimation, red and white blood counts, etc. Many diseases not local to the kidney produce haematuria, arteriosclerosis, focal infections, haemophilia, scurvy, renal infarcts and pyaemic abcesses in the kidney, poisoning by certain drugs, such as mercury and turpentine, idiosyncracies for drugs as a rule harmless, such as hexamine. Haematuria may in rare cases be nothing more than a vicarious menstruation and finally, when after strict search, no cause can be found, we may term it "essential haematuria".

JACQUES LOEB

L. KAZDAN, '27

HOW many medical students are acquainted with the name of Jacques Loeb? Yet it is they, before whom constantly looms a helplessness in understanding the fundamental principles of disease and its treatment, who will appreciate and profit most by a knowledge of his work. Not that the work of Loeb deals directly with these problems; and far distant as the solution of these may still seem to be, it is nevertheless universally admitted that his investigations into the fundamental principles of the phenomena of life may have a profound bearing upon the solution of these problems in the future. Not alone for his momentous discoveries or the avenues of new research which these have opened up, is Jacques Loeb of interest, but also for his brilliant personality, which appears to have stamped itself upon the world of science in no less a degree than did his work.

Jacques Loeb was born in Germany on April 7, 1859. He attended the Askanische Gymnasium in Berlin, and later took up Medicine, attending successively the Universities of Berlin, Munich and Strasbourg. He took his degree at Strasbourg in 1884, and passed his state examinations

there in 1885. Though he made an attempt to practise medicine, his mind was already preoccupied with research problems, and consequently he had to give it up. In connection with this, it is related how on one occasion, being apprenticed to an eye specialist, he was left to massage a patient's eyes for a few minutes. When the specialist returned half an hour later, there was Jacques, drowned in thought, still standing and rubbing away at the patient's eyes. On being wakened from his reverie, "Ach", said he, "I forgot, I was thinking about my experiment".

Though some of his work had already begun in Germany, most of it was accomplished in America. Being appointed Associate in Biology at Bryn Mawr College on his arrival in America in 1891, he was successively associated with the Universities of Chicago and California, and was finally appointed head of the Physiological section of the Rockefeller Institute for Medical Research, which position he occupied until the time of his death, on February 12, 1924. At the time of his death he was a member of numerous scientific societies throughout the world.

The perplexing problems relative to living matter early engaged the mind of Jacques Loeb. The explanations, which classical philosophy and metaphysics had to offer regarding these, were, he found, a mass of meaningless verbalism. In experimental science alone, he believed, lay the key to the elucidation of these problems.

Perhaps one of the greatest attributes of Jacques Loeb was his acuity in grasping the significance of, to correlate, to utilize for his own purpose, any new conception or discovery, no matter in what field it might be made. Primarily he was interested in the problem of Freedom of Will, and the work of Von Sachs upon the effect of light on the movements of plants, suggested a means whereby this problem might be elucidated. That not only freedom of will, but our whole inner life, and even our ethical views might be amenable to a physico-chemical explanation was the view expressed by Loeb as a result of his experiments upon the behaviour of lower animals to such agents as light, electricity, gravitation and so on. To quote from his book, "The Mechanistic Conception of Life", . . "That in the case of our inner life a physico-chemical explanation is not beyond the realm of possibility, is proved by the fact that it is already possible for us to explain cases of simple manifestations of animal instinct and will on a physico-chemical basis . . . As the most simple example we may mention the tendency of certain animals to fly or creep to the light. We are dealing in this case with a manifestation of an instinct or impulse which the animal cannot resist. It appears as if this blind instinct which these animals must follow, although it may cost them their life, might be explained by the same law of Bunsen and Roscoe, which explains the photochemical effects of inanimate nature."

In the introduction to his book "The Organism as a Whole", Loeb deplores the fact that while single processes occurring in the animal body, as for example: digestion, metabolism, heat production, etc., are universally explained upon a physico-chemical basis, yet when the actions of the organism as a whole are concerned, they are regarded as the expression of non-physical agencies.

His work on animal tropisms, as the above studies have been termed,

laid down the foundations of experimental psychology. He analyzed the factors on which different tropisms depended. Heliotropism, for instance, depends on concentration of H_2CO_3, temperature, age, and so on. That heliotropism may be a factor in abnormal psychology, because of the action of acid or secretions from reproductive glands which cause the organism to respond in a manner which is not its normal behaviour, was the view expressed by Loeb.

His predominating idea that all life phenomena are determined by physico-chemical processes led Loeb into various other fields of absorbingly interesting research. Only a few of these can be given the briefest outline.

In Experimental Morphology his work revealed the fundamental laws of growth and regeneration. "Growth takes place in certain cells when certain substances in the circulation can collect there. These substances are attracted to certain spots and this prevents them from acting on other parts of the organism. If such parts are isolated the substance can no longer flow away from these parts, and the parts begin to grow . . . Regeneration thus need no longer be explained on the assumption of a directing force which has to elaborate the isolated parts into a whole".

Loeb's studies in artificial parthenogenesis were epoch-making and strikingly show his creative genius. Step by step, leaving nothing undone, solving every seemingly insolvable problem by simple and decisive experiments, he reduced the mechanism of fertilization to almost its ultimate principles. The belief which was prevalent, that the fusion of egg and sperm nucleus is essential to the development of the egg, was disproved by the experimentation of Loeb. He furnished proof that the activating effect of the spermatozoon upon the egg can be replaced by a purely physico-chemical agency. The essential feature in the activation of the egg by the sperm was found to be the formation of a membrane around the egg, induced by the action of a lysin in the spermatozoon upon the cortical layer of the egg. This could equally well be brought about by treating the egg with various cytalytic substances, such as acetic, butyric, or caprylic acids, etc. That, however, the egg should develop into a normal larva, a second corrective treatment with a hypertonic solution or lack of oxygen was found necessary. In this way the eggs of sea urchins, and by other means the eggs of frogs, were developed to the adult stage of the animals. The possibility of some day being able to accomplish this in warm-blooded animals was expressed by Loeb.

His studies on the Antagonistic Action of Salts reveal the extent to which physico-chemical science has benefited from the work of Loeb. Having gained an insight into the action of salts in blood and sea-water, he proceeded to investigate the role which these might play in the life of the cell. His work in this direction is summarized as follows: . . . "The role of salts in the preservation of life consists in the 'tanning' effect which they have upon the surface films of the cells, whereby these films acquire those physical qualities and comparative impermeability without which the cell cannot exist . . . they do not furnish energy."

Regarding the definitions of Life and Death, Loeb arrived at the following conclusions: "The difference between life and death is not one be-

tween living and dead molecules, but more likely between the excess of synthetic over hydrolytic processes". The duration of life, he concluded, was the time required for the completion of a chemical reaction or reactions. After death, due to the lack of oxygen, autolysis of the cells of the organism sets in and disintegration of the body results.

In confirmation of these conclusions his experiments on the Temperature Coefficient for the Duration of Life, are very interesting. Assisted by Northrop, he found that the fruit-fly, Drosophilla, which on the average lives only 21.5 days, could be made to live 177.5 days, provided it is kept in a chamber at lowered temperature and protected from germs of disease. This result showed that chemical reactions in the body, as outside the body, proceeded slower at lower temperatures, than at higher.

It is impossible within the compass of one article to touch further upon his work, and only a glimpse may be given of the character of the man himself.

Loeb was an incessant worker. Before he was out of bed he would be planning experiments, and after a long day in the laboratory he would spend the evening reading. In thirty years he practically never took a vacation. His life became more and more his science. Yet this never meant a narrowing of his interests. He read enormously in fields of science outside of his own, and took an interest in every realm of human thought. He could converse on equal terms with men like Einstein and Sir Ernest Rutherford. He could meet on their own ground the world's foremost chemists, astronomers, botanists, biologists and political economists.

Probably no man shrank more from personal publicity than Jacques Loeb. If he had his way the lay public might never so much as have heard of his name. To a friend who asked his permission to write his biography he replied, "As long as I am able to do work I want to put my mind exclusively on my problems and not the possible share I may or may not have had in solving them". He was a disciple of Diderot, whom Morley pictures as "one of those simple, disinterested and sterling workers, to whom their own personality is as nothing in the presence of vast subjects that engage the thoughts of their life".

There is no better index to the greatness of this man than the regard in which he was, and is, held by his associates and those who knew him. "The most dramatic feature of Loeb's career", writes R. L. Duffus, "lies in the contrast of his personality and the popular conception of what a mechanistic biologist ought to be. Here was a man who believed that living organisms were machines, and that their reactions could be explained according to the same principles which are used by chemists and physicists; who declared that consciousness and free will are illusions; who reduced the Lord of Creation to the status of a chemical solution bubbling in a test tube, and who recognized nowhere in the universe a purpose or a God. Yet Jacques Loeb maintained it with the pure selfless devotion of a crusader, sacrificed every worldly interest to it, upheld it with almost saintly passion, not as a curse of disillusionment brought upon humanity for its sins, but as a gateway of escape into happiness and freedom. He was a passionate believer in the goodness of

humanity, a hater of war, a democrat, who never for a moment conceded the superiority of one race over any other; a man so kindly that in the days before anaesthesia was used in laboratory animals, he gave up such experiments, valuable though they might have been in his researches. A lover of natural beauty, a seeker after classic simplicity, a companion with a quality so witty, so penetrating and so magnetic that despite his modesty, he became the natural centre of any group he was in." He was, above all, a man of whom none of his friends can speak for five minutes without using the adjective "lovable".

FUNCTIONAL HEART DISEASE IN CHILDREN

MANSFIELD G. LEVY, M.D.*
Buffalo, N.Y.

The term "heart disease" covers many conditions. It includes congenital heart disease, endocarditis, pericarditis, and myocarditis, in their various forms and phases, and especially in children, functional affections.

In this brief paper, only functional affections will be considered. Let us first understand what is meant by this term—functional. In recent years, there has been a tendency to include under this term all conditions where a symptom-complex was found to obtain, including a soft systolic murmur, usually over the base, and associated with none of the more common signs of true cardiac disease, such as hypertrophy, dilatation, or accentuation of the second pulmonic sound. I feel that the above definition is incomplete. Jacobi has well stated that "the diagnosis of deranged function in any organ is only a makeshift, and justifiable only so long as we are ignorant of the physical cause of the derangement." A functional heart murmur is one whose anatomical or underlying cause we do not know. That is why a skilled diagnostician may recognize fewer functional murmurs than one who will not diagnose a heart disease unless he has all the symptoms, including enlargement. It has therefore been the purpose of cardiologists to eliminate as many as possible of these so-called functional cases, assigning them to well-founded organic causes. Thus we may see that certain symptoms formerly thought to be functional, are in reality due to a change in the shape and position of the heart. In the same manner, the cardiac arrhythmias have been quite recently analysed, and classified, with the result that a distinctly demonstrable organic basis has been found for the so-called functional irregularities.

Although at the present time most text-books still adhere to the term "functional murmur," I prefer to discard it and substitute "atonic," "haemic," "cardiopulmonary," or accidental, depending on whatever I believe to be the underlying cause.

The majority of these so-called functional murmurs are cardio-pulmonary in origin. This murmur originates outside the heart; hence,

*Reprinted from Archives of Paediatrics, Vol. XLII, No. 9.

change in the posture of the child, by altering the relation of the heart to the surrounding viscera, may alter the character of the murmur, or even cause its complete disappearance.

Schlieps uses the term "atonic" for the murmur heard over the hearts of asthenic children. This murmur is usually heard over the apex, differing in this respect from the cardiopulmonary murmur heard over the base. The atonic murmur is supposed to be due to a relative insufficiency of the mitral valve following a loss of tone of the heart muscle.

In making the differential diagnosis between these so-called functional, and organic murmurs, the following points are of paramount importance:

1. The nutrition of the child.
2. The type of child (is the child asthenic?).
3. The murmur itself. Here we must consider the character of the murmur; its time of occurrence; location over the precordium; influence of change of posture.
4. The shape of the heart shadow as seen under the fluoroscope. Here the signs of the mitral shape, or signs of ventricular hypertrophy are in favour of organic disease.
5. Presence or absence of other signs of congenital, or acquired heart disease.
6. X-ray and the electro-cardiograph are very commonly of decided value in their points of differentiation.
7. History of rheumatism, tonsilitis, chorea, or other infection. Lues rarely, if ever, causes organic heart disease in children, hence the relatively small percentage of aortic lesions in children.

While I believe that functional conditions do exist, and that many of the truly organic cases are so moderate that they functionate as inorganic, I also feel that in our anxiety to be fair with our patients, we often say a given case is not organic when we have failed to make a sufficiently painstaking examination. Nor do I mean to imply that because a given case is organic, the patient's life should be radically altered, or that encumbrances be placed upon him to make him worthless to society.

It is to be noted, as stated above, that there is a great similarity between the cardiac signs of both functional and true disease. We meet with not only murmurs, but also irregularities, dilatation and hypertrophy in both. It shall be my aim to make the differentiation clear. In some cases this can be positively demonstrated; in others, one can only indicate certain aids. I shall take occasion to report some interesting though not unusual findings. They have been overlooked in the past because of want of instruments of precision. While this paper is not concerned with a study of electro-cardiography, it often becomes necessary to have a given case traced. In this way it will be shown that electro-cardiography has an indispensable place in paediatrics.

The conditions most generally spoken of as functional, and which many times have been found to be organic are sinus arrhythmia, certain cases of heart block, including some forms of bradycardia, and the systolic murmurs heard over the base in the pulmonic area.

A simple sinus arrhythmia consists of an alternating waxing and

waning of the heart rate. Groups of slower cycles alternate with groups of more rapid ones in the electro-cardiogram. The rapidity of the alternations depends upon the rapidity of respiration. The individual complexes in the electro-cardiogram and their relations to one another, are unchanged. But let me add a word of warning. Not all the arrhythmias irregularities of the heart as heard by the stethoscope or felt at the pulse are simple sinus arrhythmias.

There are organic irregularities that may be explained by the presence of a myocarditis or a pericarditis; in fact one authority argues that were it possible to autopsy cases showing irregularity, many of them, if not most of them, would show evidence of a true myocarditis. Some of these irregularities, especially those associated with rheumatism, originate in a myocarditis that exists because of the presence of Aschoff bodies. Frankel reports finding these nodules in 17 out of 20 cases autopsied by him. In this connection, I have in mind a little girl seen at the Goodyear Convalescent Home, who had no demonstrable heart lesion clinically; but there was an irregularity of the pulse. X-ray findings were negative, but tracings gave proof that the gir had a definite myocarditis evidenced by the presence of premature contractions. The electro-cardiogram is indispensable for accurate diagnosis of these abnormalities.

Occasionally we meet with cases in which an abnormally slow heart attracts attention. This may be due to an infection associated with rheumatism, diphtheria, influenza, or other infection. In most of these cases we recognize the slowing as a symptom of some definite affection, and are not likely to refer it to any disease of the heart. However, when a persistent slowing of the heart is observed in early life, with or without obvious cause, then suspicion must be aroused of the existence of true heart disease. The more special form of slow pulse which is associated with heart block is not common in children. A few isolated cases of heart block in children in association with diphtheria have been reported. Flemming and Kennedy have reported one in a boy of 10, where there was a ventricular rate of 46-54, and an auricular rate of 100-110. Necropsy showed a marked myocarditis. Recently, Levine of Boston reported a series of blocks, where the ventricular rate was between 50 and 60. How is one to recognize a blocking of this kind if not looked for by an electro-cardiograph tracing? We might call these cases simple bradycardias, looking for the explanation of their existence outside the heart.

Simple systolic murmurs, heard most commonly over the pulmonary area, may or may not be functional. I have heard many such, which at first thought appeared most certainly to be functional. Especially might I have been reassured that such was the case, if there had not been any of the signs associated with congenital pulmonary stenosis. Yet, what would you say if I told you that many of these same children showed R.V.P. and considerable amplitude of the ventricular complex which is conclusive of congenital disease? Likewise, I have seen many children who had simple soft murmurs over the apex, and who appeared perfectly normal and healthy otherwise, yet when these cases were X-rayed and measured, they showed definite enlargement. Occasionally

some showed L.V.P., others when viewed fluoroscopically, even showed typical mitral configuration.

In closing, may I state that the purpose of this paper is to awaken a spirit of thorough investigation of each and every case that comes to us with heart murmurs. Not only will we obtain accuracy in our diagnoses, but such investigation will show how much more uncommon functional conditions are, than has previously been acknowledged.*

REFERENCES

Trans. Amer. Climatol. Assn. (1899), p. 41.
Schlieps. Jahrb. f. Kinderh., LXXVI (1912), p. 247.
Sutherland. The Heart in Early Life, p. 136.
Frankel. Diseases of the Heart, p. 287.

PROTEIN MILK IN INFANT FEEDING*

GORDON C. KELLY, '26.

Presented before the Paediatric Reading Club, Dec. 19, 1925.

In the last fifteen years protein milk has come into general use in all paediatric hospitals in connection with the artificial feeding of infants. Unfortunately, however, it is not as yet employed by general practitioners nearly as often as its use is indicated.

It has long been known that there is a difference in the tolerance of infants for cow's and for mother's milk. Finkelstein showed that milk cannot be considered as an entity, but that the effect of each ingredient must be studied separately. He fed infants on cow's milk casein and mother's milk whey, with good results; in another series he fed infants on cow's milk whey and mother's milk casein, and found that the intestinal disturbances encountered on ordinary milk dilutions appeared. He found, in part, that,—

(1) Casein is not harmful. Protein tolerance is greater than fat or carbohydrate tolerance. In fact, in the presence of a high percentage of casein, the carbohydrate tolerance is raised.

(2) Casein undergoes putrefaction. Ammonia is formed, so that alkalinity results in the colon, which directly opposes the pathological acid fermentations of disturbed carbohydrate and fat digestion.

(3) Fat is sometimes harmful, *e.g.,* in the presence of excess carbohydrate fermentation, when it undergoes harmful catabolic changes. CaO from calcium caseinate forms with fatty acids lime soaps, so that in two days, sometimes, firm alkaline soap stools are formed.

(4) The whey salts of cow's milk differ greatly from those of mother's milk, and influence digestive functions accordingly. Mother's milk has 0.2 per cent. of salts, cow's milk 0.7 per cent.

It has been demonstrated that the bacterial flora of the intestinal tract of infants is determined by the variety of food given. The pathological process in most infantile diarrhoeas is due to the acid

*Many slides were shown to illustrate and prove statements made by the author.

fermentations brought about by saccharolytic bacteria, in conjunction with overtaxed function of the digestive mucosa. An abnormal flora arises in the acid medium produced, which aids in splitting up the fat and sugar into low fatty acids, (acetic, butyric, succinic, valeranic, etc.) which further irritate the mucosa, and result in the production of mucus, increased peristalsis, and diarrhoea. This wastes the soluble sodium and potassium salts and produces a relative acidosis, which adds to the toxemia. There is also a large water loss, depending on the water-binding stability of the infant, and on its previous feeding. This pathological process continuing, digestion becomes suspended, and finally food has a paradoxical effect, not nourishing the child, but increasing the colitis and diarrhoea. Thus is established a vicious circle which can end only in fatality unless broken.

Finkelstein foresaw from his work the need of a food which both suspends abnormal fermentations, and tends to prevent dehydration, at the same time providing sufficient nutrition to tide the infant over until the intestinal mucosa could heal and recover its functions. To fill this need he prepared "eiweiss milch" from whole milk, rennin, and water. Many modifications have since taken place, as the result of much work on the subject; and in the Hospital for Sick Children, Toronto, protein milk is now made by the following method:—

Directions for Making Protein Milk—From the Dairy, H.S.C., Toronto.

One quart (32 oz.) of 4% raw milk is taken. Two teaspoonfuls of essence of pepsin is added (or a junket tablet). Warm 10 min. to not more than 140°F. Keep at this temperature for 10 min. (Too great heat will cause the curd to become tough.) Let it stand at room temp. for ½ hour. Then separate the curd from the whey, place the curd in a cheese-cloth bag, and allow it to drip, in a refrigerator. This takes from 2 to 4 hours, but in large amounts the dripping may be allowed to proceed overnight. The curd is then put through a potato-ricer, then worked through a fine wire sieve (30 wires to the inch) with a broad wooden pestle, at the same time adding one pint of a fat-free lactic acid milk, (or buttermilk if no lactic acid milk is available). Finally add cold boiled water up to 1 quart, and 1 grain of saccharin to every finished quart of the protein milk.

Precautions to be Observed in Making Protein Milk.

1. (*a*) Fat content of protein milk may be reduced by churning some of it into butter. To prevent this, all ingredients,—curd, buttermilk, and water,—should be absolutely cold when being worked through the sieve. A warm temperature helps in separation of cream into milk and butter.

(*b*) The masher or pestle should not be worked in rotary action. Pressure should be used each time to get the curd through with the minimum amount of churning of the milk.

2. (*a*) As the curd is being put through the sieve it must be kept dripping with buttermilk. If pressed through fairly dry it clots on the under side of the sieve.

(*b*) If more liquid is required to keep the curd wet, one uses some of the protein milk already sieved.

This seems a long and difficult procedure, and many modifications have been tried, always with more or less failure, so it would seem advisable to adhere rigidly to the directions. The technique was adopted only after four years of careful experimentation with various procedures.

Average Composition of Protein Milk, made from 4% milk.

Moisture %	Ash %	Fat %	Sugar %	Protein %	Calories
93.96	0.55	1.59	1.60	N x 6.25% 3.16	per oz. 10

Characteristics of Protein Milk are:

(1) Its dilution, yet concentrated food value.

(2) The small content of lactose and soluble whey salts.

(3) The increased amount of casein present, which permits early liberal feeding. The casein exercises its therapeutic action in preventing acid fermentation, and thus assists in the repair of damaged mucosa. It soon permits feeding of a higher percentage of carbohydrates and fats, which quickly raises the tolerance towards cow's milk. The protein is also rich in calcium and magnesium salts, the insoluble salts, to which is ascribed its value in producing formed stools.

(4) The low fat content.

(5) Finely divided curds.

(6) The higher acidity than in ordinary cow's milk mixtures. Gastric acidity is necessary for proper protein and fat digestion. It is difficult to bring cow's milk up to this acidity because of its larger concentration of buffer salts. Therefore less complete digestion results with cow's milk.

(7) Its predigestion with essence of pepsin (or rennin, or junket).

Thus protein milk is a soured milk product, a mixture of cottage cheese and buttermilk. It is a high protein food with finely divided curds, a moderate fat content, and a low milk sugar content. It has a rather sour, pungent taste, which is somewhat corrected by the addition of the saccharin, so that infants do not greatly object to it.

Indications for Use.

1. In all cases of diarrhoea, particularly all cases of fermentative diarrhoea. In infectious diarrhoea either protein milk, or lactic acid milk plus added corn syrup, may be used.

2. In dyspepsia in bottle-fed babies, and in many cases of dyspepsia in breast-fed babies, right from the start.

3. In chronic intestinal indigestion in older children.

4. In marasmus, atrophy, or decomposition, during the reparative stage.

5. As a complemental or exclusive "prophylactic" food for newborn, premature, and other infants needing a high protein intake.

6. In cases of parenteral infections to counteract loss of appetite, also to avoid gastro-intestinal upsets.

7. Useful in cases with low fat tolerance.

8. Some claim that, though not indicated, it may be used for healthy babies. Here the cost, averaging one cent per ounce of full strength mixture, is an objection.

Technique of Administration.

It is usually taken readily, except in children over one year, when in order to get them to take it, it is necessary to starve them. Occasionally it must be given by stomach tube. When using, the milk must not be heated too rapidly, nor above 90°F., otherwise tough masses of casein are likely to form. A large hole in the nipple is necessary. The bottle must be shaken every few minutes during feeding, as the curds tend to settle.

In severe cases, with vomiting or intoxication, and numerous stools daily, there is a preliminary period of starvation, until the toxaemia passes off. During this time glucose solution 5% by mouth, or 10% by nasal drip is administered, to supply fluids. Interstitial and intravenous injections of 10% glucose are given as required to overcome dehydration. Then one feeds ½ or 1/3 strength protein milk, depending upon age, and offers 3 oz. per lb. body weight (but not exceeding 40 oz. per day) on account of the necessity of getting fluid into the infant. Then as the frequency of stools decreases, the strength of the feeding is slowly and gradually increased up to full strength. When formed stools occur, carbohydrate is added gradually to the feeding up to the caloric requirements, at the same time being used to govern the consistency of the stool formation. Dextrimaltose is preferable, as it ferments more slowly than most other sugars. Never use lactose. In mild cases there need be no preliminary starvation, and the stage of full-strength feeding is reached in a few days.

Duration of Administration.

There is usually some loss of weight during the first few days, as the caloric intake is below the requirement. In 4 to 8 days the stools usually become characteristic of protein milk diet—regular, 1 or 2 daily, large, light yellow, and putty-like; rather dry, almost odourless, and free from unmixed particles or mucus.

The corrective period is naturally divided into three stages,—

(1) That in which protein milk alone is given. This averages 6 to 8 days, but occasionally may last 3 weeks.

(2) That in which protein milk plus dextrimaltose is given. The carbohydrate is added gradually as soon as formed stools occur, up to caloric requirements. If carbohydrate tolerance is low, cooked barley flour, which is even less laxative than dextrimaltose, may be used. This stage averages 2 to 4 weeks, but has often been continued successfully for 8 months or more, with consistent gain in weight and strength.

(3) That in which protein milk plus some plain or other milk mixtures is given. The latter may be added either gradually or suddenly, and there is seldom a necessity to return to protein milk feeding. Often protein milk is used as a supplement to breast-milk

feeding. In the corrective stage, at first calories reach a low total, and a gain in weight is not to be expected, rather a small loss. When other food is added, the high protein content assists assimilation, and a gain is often made, but this cannot be expected if the infant is fed less than 35 calories per lb. body weight; yet in many cases in the Hospital for Sick Children, Toronto, such a gain has been reported.

Failures are due to

1. Poor preparation of the protein milk.
2. Inanition due to insufficient feeding of protein milk.
3. Inanition due to delayed or insufficient addition of carbohydrate.
4. Damage due to cutting down on the food, if any symptoms reappear for a time, in cases which responded well at the start.

Dr. Alan Brown and co-workers have reported a series of 100 cases, ranging in age from birth to over one year, which had been fed on breast-milk, milk mixtures, or proprietary foods, and which were admitted in conditions ranging from "not acutely ill" to "moribund." Of these, 19 died, 1 did not improve, 3 improved, and 77 were cured of all symptoms. The average stay in the ward was from 21 days for the younger infants, to 34 days for the older infants.

The great disadvantages of the liquid protein milk are

(1) Difficulty of preparation.
(2) Its perishable nature.
(3) Some trouble experienced in feeding it to infants.

These difficulties almost wholly excluded it from general use by physicians, and so a powdered product was sought. After much experimentation, trials, and failures to obtain a suitable and satisfactory product, there are now several powdered protein milk mixtures on the market, for each of which certain advantages are claimed by their manufacturers. The powder used in the Hospital for Sick Children, Toronto, is largely one prepared through co-operation with the Canada Milk Products Co. (C.M.P.). Its ingredients are

(1) Pasteurized fat-free milk.
(2) Casein.
(3) Cream.
(4) Lactic Acid Starter.

These are mixed in proper proportions in an enamel-lined tank equipped with a propeller. The mixture is then rapidly dried by spraying it through a jet of hot air. Thus it is instantly transformed into a powder having a moisture content of not less than 2%. At no point does the temperature rise above that of pasteurization, except during the removal of moisture, which requires only a fraction of a second. Thus the living Streptococcus Lacticus is still present in C.M.P. powdered protein milk, due to the process of manufacture.

The chief differences from the liquid preparation are

(1) The lactose content is twice as great in the powder.
(2) The acidity of the powder is a little more than one half that of the liquid.

There are no serious clinical objections to these.

The powder is a uniform product, and when mixed will look like milk, though it tends to settle and then merely requires shaking to mix again. The standard liquid protein milk is made by adding 1 part by weight of powder to 11 parts by weight of water, 1 oz. of powder being equal to 5 level teaspoonfuls of powder. The cost is approximately one cent per ounce of full strength liquid.

Analysis of Protein Milk.

	Powder (C.M.P.)	Standard Liquid Prep'n
Protein	38%	3.16%
Fat	27%	2.25%
Lactose	24%	2.00%
Ash	5%	0.42%
Moisture	3%	91.92%
Lactic Acid	3%	
	100%	100%
Total Acidity	5.75%	0.48%
Calories per ounce	143.	12.
pH		3.8

Composition of several protein milk powders, compared with *Whole Cow's Milk,* and *Mother's Milk.*

Product	Powdered	Fat%	Lactose %	Protein%	pH	Calories per oz.	Av. cost per quart
Cow's Milk	—	4.0	4.5	3.5	7.—	21	$0.12—0.18
Mother's Milk	—	3.55	6.303	1.944	7.+	21	3.00
Finklestein's	—	2.5	1.5	3.0	7.—	12.25	0.38
Larosan-Roche	P	1.7	2.2	3.4	7.	12.42	0.37
Hoobler's	P	1.75	2.0	2.8			
Mead's Casec	P	2.0	2.0	3.0	6.8	12	0.20R, 0.17Wh
Mead's Powdered Protein Milk	P	2.58	1.58	3.08	5.	12.	0.125
Hoos' Albumin Milk	P	2.5	1.5	3.00	5.5	11.9	0.42R, 0.30Wh
Protolac	P	0.10	2.63	3.54	6.8	4.23	0.25
Merrell-Soule	P	2.25	2.0	3.16	3.8	12.6	0.35
Acidulated Protein S.M.A.	P	2.2	2.8	3.5	4.6	15.	
Walker-Gordon's	—	2.5	2.0	3.0		15.	0.60

R=retail. Wh=wholesale.

All figures originally obtained from the respective companies.

It should be noted that larosan and casec are simply soluble protein to add to ordinary milk mixtures, and are not used separately. Mead's powder is dried and does not contain living organisms. C.M.P. and Merrell-Soule are the same, and contain living lactic acid organisms.

Chief Advantages of Protein Milk Powder (C. M. P.)

(1) It may be kept for several weeks and possibly months.

(2) It may be mixed with either cold water or water up to 110°F. without any separation of the curd.

(3) It flows through the nipple in a manner much like whole milk dilutions, thus obviating the necessity of frequent shaking of the bottle, or removing the nipple.

On the whole, clinical results have been most encouraging in the Hospital for Sick Children, Toronto, following indicated and proper usage of protein milk.

It has been noted, in addition, that the infants generally reacted more rapidly (*i.e.*, the stools became "formed" in a shorter time), and made more consistent gain in weight with the use of the powdered product. In this hospital protein milk powder mixtures are used almost always, where protein milk is prescribed, because of the ease in making it up. Therefore its use is stressed rather than that of the liquid protein.

The writer desires to express appreciation for many helpful suggestions from Dr. Drake, of the H.S.C., Toronto, in the writing of this paper.

BIBLIOGRAPHY

1. Brown, Spohn, and MacLachlan—"Protein Milk—Its Composition, Preparation and Application in the Treatment of Digestive Disturbances." Can. Med. Assoc. Journ.—June, 1918.

2. Brown and MacLachlan—"Protein Milk Powder." Can. Med. Assoc. Journ.—June, 1919.

3. V. A. Blenkle, M.D.—"Protein Milks in Infant Feeding." Archives of Paediatrics—Nov., 1925.

ON THE PATHOLOGY OF BRIGHT'S DISEASE (*Continued*)

B. I. Johnstone, M.B.
Late Fellow in Pathology, University of Toronto

Chronic Bright's Disease

Under this term we can include a large group of kidney lesions resulting in renal insufficiency. It is obvious, however, that no sharp distinction can be made at present, either clinically or pathologically, between the vast majority of cases.

The pathology may be discussed on a plan similar to that followed in the acute phases.

I. Degenerations

We have seen how various poisons acting on the parenchyma of the kidney will produce a nephrosis. In the milder cases, as seen in association with the acute infectious diseases, with the removal of the toxin, we have complete recovery, no scar remaining in the kidney as a result of the lesion.

In the more severe cases where we have extensive degeneration involving the whole secretory unit, even to necrosis of the cells and the deposition of calcium salts owing to the remarkable ability of the tubules to regenerate, if the condition be purely degenerative and the patient recovers from the acute attack, we will have complete recovery. Volhard and Fahr describe healing here by scar formation. It is conceivable that with complete degeneration of the epithelium the tubule might collapse, the glomerulus atrophy and scar result. If this does occur it is very

rare, and in such cases we cannot dissociate a superimposed insult due to lowered resistance resulting in inflammation and subsequent scar formation.

Thus in the acute uncomplicated cases of nephroses in which the causative agent is removed, no evidence finally remains of the preceding acute lesion.

If the poison or toxin continues to act over a long period of time, chronic degeneration occurs and we have a condition of chronic nephrosis. We will therefore review such processes in the kidney.

Fatty Degeneration:

Fat is not visible in the normal renal cells or else only in exceedingly small amounts. Its appearance, therefore, is of considerable importance, because its signifies a definite retrograde change. Fatty degeneration cannot be considered as producing a distinct type of kidney lesion, as it is merely associated with, or a consequence of some other more important condition.

Fatty degeneration quite commonly accompanies such general systemic condition as diabetes mellitus. Fat may also appear in the cells as a result of defective nutrition. Thus in anemia, fat may be present in the greatest abundance, diffusely scattered throughout the kidney, especially in the loops of Henle and the convoluted tubules. Again, when the nutrition of the kidney is affected as the result of lesions in the blood vessels, fat may appear in localized areas of the epithelium of the tubules involved. Fat is also commonly seen in the so-called large white kidney where degeneration is taking place secondarily to glomerular changes. In such cases the fat may be in combination with cholesterol and then does not give the characteristic red stain with Sudan III.

Amyloid Deposit in the Kidney:

Chronic degeneration in the kidney also occurs in association with the deposition of amyloid. Traube was the first to separate the amyloid kidney from nephritis. It is quite true that the presence of amyloid is most frequently associated with inflammatory conditions outside of the kidney.

Amyloid is a colourless, homogeneous, translucent material, most probably a compound of chondroitin sulphuric acid and a protein. Mallory assumes it to be manufactured from the blood by abnormal cell activity on the part of fibroblasts. It most commonly occurs in the liver, spleen and kidneys in connection with severe wasting diseases, particularly in prolonged tuberculosis, suppuration and ulceration, especially of the bones.

In the kidney it is usually formed around the glomerular capillaries and the smaller arterioles. Later it may appear around the veins and tubules. The presence of amyloid leads to narrowing of the lumen and finally occlusion of the vessel. Finally, the glomerular tuft or the whole malpighian body may be represented by a mass of amyloid. By interfering with the nutrition of the epithelium, and also by mechanical pressure, extensive degeneration of the parenchyma may result. The

epithelial cells undergo a peculiar colloid type of degeneration. Amyloid also most probably interferes directly with normal secretion.

In the gross, the kidney in the early stages may be double its normal size. It is pale in colour and frequently has a rough, finely granular surface, giving the impression of a cat's tongue when the finger is drawn over the cut surface. The increase in size is not so much due to the presence of amyloid as to the swollen parenchyma. In the late stages when the tubules atrophy, the kidney may be small and shrunken and weigh less than half its normal size. The presence of amyloid is readily demonstrated in the gross by dropping a section in a solution of iodine. The amyloid stands out a deep brown.

II. Chronic Nephritis

If we follow our cases of acute nephritis, we find that a certain number get well, leaving varying amounts of scar tissue in the kidney. Such cases, though completely without clinical symptoms, have not completely recovered, in that it is generally recognized that the kidney once attacked by nephritis is analogous to the lung in lobar pneumonia, or the heart valve in endocarditis, in that it seems to be more susceptible to a recurrence of the inflammation. In a second group following the abatement of the initial acute symptoms the patient does not get well, but passes through the well-known subacute and chronic stages.

Those cases of chronic nephritis, however, in which we can obtain a history of a former acute attack, make up only a very small percentage of the total number of cases. By far the greater number are chronic from the first, or at least not sufficiently acute to drive them to a doctor, and the lesion thus passes unnoticed and continues to develop insidiously. By the time the evidence of renal insufficiency is observed, extensive changes may have taken place in the kidney. It is generally assumed that the etiological factors concerned in acute nephritis are also responsible for the development of these chronic cases. The insult being less or the resistance greater, a less acute reaction is produced.

A vast amount of work on experimental animals has been done in an attempt to produce kidney lesions resembling those found in man. A great variety of substances have been used for this purpose. Among the commoner poisons utilized are cantharidin, diphtheria toxin, uranium nitrate, snake venom, arsenic, salts of mercury, phosphorus, aloin, sulphuric acid, chromium, etc. As early as 1884 Cornil and Brault were using cantharidin in an attempt to produce typical lesions in experimental animals. Since that time a host of workers have invaded this field. Important contributions have been made by Welch, Lyon, Ophüls, Pearce, Dickson, Christian, Emerson and many others. A great deal of information regarding the pathological processes has been gained in this way, but such substances are not likely to be the cause of nephritis in many human cases.

A considerable number of observers attribute obscure cases of chronic renal disease to autointoxication. Harvey (1911) injected parahydroxyphenylethylamine, which is found in the human alimentary canal in certain conditions, into experimental rabbits, over varying periods of

time. He describes changes in the renal vessels with secondary parenchymal changes. Newburg has especially stressed the importance of excessive protein intake in the etiology of chronic nephritis. He asserts that chronic nephritis is largely caused by the combined effect of bacterial infection and the abuse of protein. Strouse and Kelman have recently made an intensive study of the effects of high protein diet on eleven hospital cases with high blood pressure. They found that raising or lowering the protein intake had no effect on the blood pressure. These experiments were carried on only for a relatively short period of time and for this reason are in no way conclusive as far as chronic nephritis is concerned.

Emerson, as do many other authors, looks upon chronic nephritis as a repetition of more or less acute attacks with periods of quiescence and healing. Others consider it a constant, progressive, insidious process. No matter whether the chronic condition has developed from an acute attack or has been chronic from the first, all forms, provided the lesion persists, tend to produce a contracted kidney. This brought about by the production and subsequent contraction of the fibrous tissue in conjunction with degeneration and nutritional atrophy in the parenchyma. However, with variation in the acuteness of the process, death may occur before this stage is reached and we may find an enlarged kidney, whereas we may have expected to find a small contracted organ. Here, even to a greater extent than in the acute forms, the inflammatory process tends to be diffuse and involve all parts of the organ.

Thus an insidious process, or a repetition of more or less acute attacks, brings about changes in the various parts of the kidney structure leading to degeneration and inflammation which finally results in fibrosis, atrophy and contraction. Briefly, the changes in the different parts of the kidney structure are as follows:

Malpighian Bodies. Certain writers claim that different glomeruli have different vulnerabilities. Greenfield and Gaskell consider the subcapsular glomeruli to be the most susceptible to change. Gaskell believes that the glomeruli with lung vasa afferentes show changes first as a result of vascular damage. In the light of the recent work of Richards and his collaborators, one could readily explain the irregularity of the distribution of the lesions in many cases by the intermittent functioning of the different glomeruli. The glomeruli affected in the acute stages, as well as those the subject of a chronic process, show varying stages of fibrosis. Connective tissue cells are readily demonstrated in the subacute and chronic stages, but whether these have developed from the proliferation of glomerular connective tissue, as Klebs and Councilman believe, or whether they have grown in from the outside, has not been definitely decided. Greenfield has shown how readily the capsular epithelium will proliferate, and this is of constant occurrence in subacute and chronic nephritis. Sometimes Bowman's capsule appears as a single layer, but more frequently it consists of several more or less homogeneous layers, separated by nuclei. There is always an associated periglomerular proliferation of fibrous tissue. At times the tuft becomes adherent to the capsule and the whole body becomes fused as a dense fibrous nodule. Other glomeruli may be

normal or show the subacute or acute reactions with cellular proliferation in the tuft, various types of thrombi, and the capsule may be thickened. The capsular spaces may contain various types and quantities of debris. Occasionally there is partial or complete atrophy of the tuft, with more or less dilatation of the capsule, forming a small cyst.

Tubules. As the glomeruli are destroyed, nutritional changes occur in the tubular epithelium. All stages of degeneration may be seen, and in the more advanced cases practically all the tubules may have disappeared, only a few hypertrophied units remaining. The cells may be swollen, coarsely granular or fatty. The epithelium may be flattened, or in other areas show hyperplasia. Other areas may show disintegrating and and desquamating epithelium, and this debris in conjunction with leucocytes is frequently seen in the lumen of the tubule. Their presence seems to depend on the degree of destruction of the tubular epithelium. There is always evidence of regeneration of tubular cells. Frequently the tubules are dilated to form small cysts.

Interstitial Tissue. An inflammatory reaction is also observed in the interstitial tissue. As a result we may have a very great increase in the connective tissue. This is due partly to the direct action of the injurious agent and indirectly to the disappearance and atrophy of the tubules. Mononuclears are called out in this non-suppurative reaction, and we find them either scattered diffusely throughout the organ or more frequently localized in small nests here and there throughout the kidney.

Blood Vessels. The blood vessels are also involved in the process and here especially the vasa, the glomerular capillaries and the small arterioles are attacked. We see proliferation of the capillary endothelium and hyaline changes in their walls. We also have periarteritis producing an increase in the adventitia, hypertrophy and hyperplasia in the media as well as proliferation in the intima associated with splitting of the internal elastic lamina. This hyperplasia and hypertrophy may gradually occlude the lumina, and when this occurs we have added to the changes described above the reaction of nutritional atrophy. Later degeneration, especially fatty, may be observed in the intima and media.

With the destruction of large numbers of the secretory units, those remaining undergo a compensatory hypertrophy in an endeavour to carry on an adequate secretion. It is estimated that there are approximately 2,000,000 glomeruli in the kidney at birth, but no new units are formed afterwards.

The gross appearances presented are exceedingly variable. On the one hand we have inflammatory exudate associated with swelling and degeneration in the parenchyma, tending to produce a large kidney, whereas with atrophy and the contraction of fibrous tissue we tend to have a small kidney. The actual gross characteristics are not an accurate guide to the histological lesions.

As the process develops, the capsule tends to become thickened and adherent to the substance of the kidney, so that when it is removed it strips with difficulty, tearing part of the organ. The surface may be coarsely or finely granular, the irregular surface being partly due to

hypertrophy of some tubules and in part to atrophy of others and the contraction of localized areas of fibrous tissue.

Frequently small cysts are seen scattered throughout the cortex often just beneath the capsule. The origin of these has been discussed. These contain a clear fluid. Small scarred areas are commonly seen in these kidneys, the result of an old infarct or arteriosclerotic atrophy.

The colour of the kidney will depend largely on the amount of blood in the organ and is not a criterion as to the pathological lesion. The peripelvic fat is usually increased. On cut section the cortex is thinned, the cortical markings are usually indistinct and frequently the cut ends of the vessels are quite prominent. This depends upon the amount of vascular change present, and the atrophy and contraction in the surrounding tissue.

We thus see that in chronic nephritis the reaction is still more diffuse than in the acute phases, and in the vast majority of cases we can only surmise which part of the structure was primarily most severely affected. If we find many glomeruli fibrosed with a correspondingly less amount of change in the other areas, the term "chronic glomerulo-nephritis" is frequently used. If the interstitial tissue is chiefly involved, we may use the term "chronic interstitial nephritis", and if degeneration in the tubules appears to be the important lesion, the condition may be descrbed as "chronic nephrosis with nephritis". Such pathological terms, however, describe only the picture observed under the microscope and are no criterion as to the various stages of the kidney through which it may have passed to reach its present condition. In fact, most writers agree that in the one individual the kidney may, and frequently does, show lesions predominating in different parts of the kidney structure at different times in the development of the lesion.

Embolic Focal Nephritis

In 1910 Löhlein differentiated between his true glomerulo-nephritis and another type occurring in subacute and chronic bacterial endocarditis. This type he called "embolische herd-nephritis", and he considered this group to have characteristic clinical and pathological features. Gaskell describes three cases and agrees with Löhlein's views. Later, Baehr described thirty-four cases of subacute bacterial endocarditis, twenty-five of which were proven to be due to streptococcus viridans. He described in detail the pathological histology of kidney lesions in these cases.

This condition is considered to be due to showers of bacteria given off from the heart valves, producing embolic infarctions in the loops of the glomerular tufts. The focal character of the lesion varies in different cases. At times only a few glomeruli appear to be involved, while at other times the lesion is quite diffuse. Löhlein was unable to culture bacteria from the glomeruli. Baehr claims to have isolated the streptococcus in a number of instances.

All cases of recurrent endocarditis do not show this lesion. Of five cases coming to autopsy in the Toronto General Hospital within the past year, three showed kidney lesions, the other two showed no kidney involvement. In one of the three the lesion was very extensive, and

histologically the kidney could not be distinguished from that of one of the so-called secondary contracted group.

Ophüls states that cases showing similar lesions have also been found in ordinary streptococcic infections and in colon bacillus sepsis. He also describes three cases of acute nephritis which were superimposed on lesions similar to those described by Löhlein and Baehr. These were the direct cause of the patient's death.

Undoubtedly, quite commonly during the course of an attack of endocarditis, evidences of renal involvement may appear in the urine, and our appreciation of this fact may prevent us from diagnosing a serious diffuse nephritis on the basis of these transitory urinary findings. However, all authors are not unanimous in considering this a distinct type of nephritis, for in these cases we do not find glomeruli alone involved. Tubules interstitial and also the vessels are affected. In the late stages, practically all writers agree—Gaskell, Baehr and Ophüls—that the kidney cannot be differentiated from the ordinary contracted organ of chronic nephritis.

It seems reasonable to assume that here, as in other cases, the process is not distinct, but rather a diffuse nephritis, with a greater tendency to involve the glomeruli in the early stages. We know that an individual with subacute bacterial endocarditis may overcome this infection. Owing to the manner of its production, with the clearing up of the heart condition, we most likely will have a cessation in the advancement of the kidney lesion. However, since we know that the kidney once involved in an inflammatory process becomes susceptible to a further attack, such an individual is more liable to a recrudescence of nephritis in later life, especially in the presence of active factors. Moreover, it has been shown that the patient may succumb to his kidney condition rather than as a result of his heart lesion. This is undoubtedly an important indication for the physician.

In this paper I have avoided mention of such extremely interesting and important factors as high blood pressure, oedema, uremia, etc. Up to the present, satisfactory correlation between clinical and pathological findings has not been made. With advance in our knowledge of pathological chemistry and physiology and with close co-operation between the clinician and the pathologist, we may hope to arrive at the solution of these obscure problems.

BIBLIOGRAPHY

Aschoff: The Pathogenesis of the Contracted Kidney. Arch. of Int. Med., 1913, 12, 723.

Adami and Nicholls: Principles of Pathologic Histology. Philadelphia and New York. 1909. Vol. 2.

Baehr: Glomerular Lesions of Subacute Bacterial Endocarditis. Jour. Exp. Med. 1912, 15, 330.

Christian: (1) Experimental Nephritis. Boston Med. and Surg. Journal, 1908, 158, 416, 452. (2) Experimental Cardio-Renal Disease. Arch. Int. Med. 1911, 463.

Cornil and Brault: Etudes sur la pathologie du rein. Paris 1884, 149.

COUNCILMAN: (1) Acute Interstitial Nephritis. Jour. Exp. Med. 1898, Vol. 3. (2) Forty-nine Cases of Acute and Subacute Nephritis. Med. and Surg. Reports, Boston City Hospital, 1897.
DELAFIELD and PRUDDEN: A Textbook of Pathology. New York, 1919.
DICKSON: A Report on the Experimental Production of Chronic Nephritis in Animals by the Use of Uranium Nitrate. Arch. Int. Med. 1909, 3, 375.
EMERSON: (1) The Acute Element in Chronic Nephritis. J.A.M.A. 1921, 77, 745. (2) An Experimental and Critical Study of the Etiology of Chronic Nephritis. Arch. Int. Med. 1908, 1, 485.
GASKELL: On the Changes in Glomeruli and Arteries in Inflammatory and Arteriosclerotic Kidney Diseases. Jour. Path. and Bact. 1911-12, 16, 287.
GREENFIELD: (1) Granular Contracted Kidney. Trans. Path. Soc. of London, 1880, 31, 157. (2) Atlas of Pathology, New Sydney Society, 1879.
HARVEY: Autointoxication and Experimental Nephritis in Rabbits. Jour. Path. and Bact. 1911-12, 16, 95.
HERRING: Jour. Path. and Bact. Edinburgh and London, 1900, 6, 459.
KLEBS: Lehrbuch der Pathologische Anatomie. 1876, 1, 44.
KELSCH and KEINER: Arch. de physiol. norm et path. Paris, 1874, 722.
LOHLEIN: Ueber die entzündlichen Veranderungen. (1) der Glomeruli der menschlichen. Nieren und ihre Bedentung für die Nephritis, Verhandl d deutsch path. Gessellsch, 1906, p. 217. (2) Pathologische Anatomie der haematogenen Nephritis. Ergebu d Allg. Path. u Path. Anat. Lubarsch u Ostertag. 1910.
LYON: Inflammatory Changes in the Kidney. Jour. Path. and Bact. 1904, 9, 400.
MULLER: Verhandlungen der deutschen pathologischen Gasellschaft. Jahrgang, 1905.
MACCALLUM: A Textbook of Pathology. Philadelphia and London, 1920.
NEWBURGH and CLARKSON: Renal Injury Produced in Rabbits by Diets containing Meats. Arch. Int. Med. 1923.
OPHULS: (1) Nephritis — Review of the Recent Literature, San Francisco. 1915. (2) Experimental Chronic Nephritis. J.A.M.A. 1907, 48, 483. (3) Some Interesting Points in Regard to Experimental Chronic Nephritis. Jour. Med. Research, 1908, 18, 497.
PEARCE: (1) An Experimental Glomerular Lesion Caused by Venom. Jour. Exp. Med. 1909. 11, 532. (2) The Production of Oedema. Arch. Int. Med. 1909, 3, 422.
RICHARDS and WEARN: Amer. Jour. Phy. 1922.
SAUNDBY: The Histology of the Granular Kidney. Trans. Path. Soc. London, 1880, 31, 148.
TYTLER and RYLE: Quart. Jour. Med. 1917-18, 11, 112.
VOLHARD and FAHR: Die Bright'sche Nierenkrankheiten. Berlin, 1914.
WEIGERT: Samml klin Votr., Leipzig, 1879.
WELCH: Trans. Assoc. Amer. Phys., 1886, 1, 171.
ZIEGLER: Deutsch Arch. klin Med. 1879, 25, 586.

SOME GREAT ARTISTS AND MEDICAL ILLUSTRATING

GORDON S. FOULDS, M.B., M.S.

St. Michael's Hospital, Toronto

ART and Medicine may seem to have very little in common except their antiquity, for while Art was making noble strides toward the attainment of its ideal, Medicine was "lagging behind century after century, wasting precious years over idle superstitions or in gnawing the

dry bones of ancient dogmas". There is no reliable evidence as to the utilization of Art by the Physician prior to the fourteenth century. Ancient teachers, as Galen and Hippocrates, apparently made no use of drawings to illustrate their writings. Aristotle, in his history of the animal world, refers to paradigmata, which probably represented parts of animal rather than of human bodies. Herophilus and Erasistratus dissected human bodies, but one finds no record of any illustrations having been made.

Artists had been far more profound in their observations than physicians. There is nothing left to be desired in the relics of Greek Art, such as those in the Elgin Collection in the British Museum, which are attributed to the chisel of Pheidios (died B.C. 432), or in such later works as the "Laocoon" and the "Dying Gladiator". In all there is a perfection of anatomic as well as sculptural conception.

With this reference to Medicine and Art in the more remote times, let us now consider them from the period of the revival of anatomic study in Mediaeval Europe to our own times. According to Anderson, the stages of advancement of Art in its relationship to Medicine may be classified as follows:—

1. The period of accredited utilization of diagrams in medical teaching, nearly coinciding with the fourteenth century.
2. The period of artistic and schematic anatomy, extending from the early part of the fifteenth to the beginning of the sixteenth centuries.
3. The period of accurate and artistic illustrations of medical scientific writings by wood cuts, reaching to the middle of the sixteenth century.
4. The period of artistic engraving on copper, which terminated in the second decade of the nineteenth century.
5. The modern period of uneclipsed technical resources in reproduction, and of painstaking efforts to secure clearness in this reproduction of accurate illustrations.

According to Guy de Chauliac, Henri de Mondeville, a physician and chief surgeon to Philip the Fair of France (1285-1314), taught anatomy by the use of thirteen illustrations. This seems to be the first reference to teaching after this manner.

The first great medical illustrator, and an outstanding man of his time, was Leonardo da Vinci, the painter, sculptor, architect, scientist, poet and musician. Leonardo was born in 1452 in the castle of Vinci in the valley of the Arno. For four years he studied as a pupil of Andrea Verocchio in Florence. He was first among artists to become associated with a physician or anatomist for the mutual benefit of both. He associated himself with Marc' Antonio della Torre, a physician born in 1473, and educated in Padua in Medicine and liberal Arts. Leonardo worked with Marc' Antonio and made numerous drawings for an anatomical treatise, which was never published because of the latter's death in 1506 or 1512. There are thirteen volumes of drawings left by Leonardo. Twelve of these are in the Ambrosian library in Milan and the thirteenth is in King George's private library in Windsor Castle. This volume contains

234-235 sheets with 779 sketches, including portraits, caricatures, single figures, compositions, riding, fencing and tournament scenes, horses and other animals, flowers, illustrations pertaining to rifle practice, optics, hydraulics and mechanics. Besides these there are a large number of very exact drawings of anatomic subjects. One of the drawings shows the semilunar valves of the aorta in a variety of positions, so as to show their descriptive anatomy and their physiologic action. He made experiments on pigs about to be slaughtered by inserting a "piercer" from a wine cask into the heart and then observed the propelling power of the heart. He also noted that the heart shortens as it empties. Though this was long before the time of Fabricius and Harvey, he could hardly have missed the significance of his observations—the circulation of the blood. His most famous works are "The Mona Lisa", "The Lord's Supper" and the "Equestrian Combat".

In 1474, when Leonardo was twenty-two years old, Michael Angelo Buonarroti was born in Caprese. He studied anatomy under the direction of Realdus Columbus, but he was primarily a sculptor and his statues, "David" and "The Captives", are wonderful evidence of his anatomical observation. He was, like Leonardo, an architect as well as a sculptor, painter, engineer and poet. The ceiling of the Sistine Chapel is probably his greatest work. Pope Julius the Second asked him to build him a mausoleum, and this lead to the building of St. Peter's in Rome, of which Michael Angelo was the architect.

Next we must consider Raphael Santi, the most perfect painter of modern times, who was also an architect. He was born in Urbino on March 28, 1483, on Good Friday, and died in Rome on April 6, 1520, also on Good Friday. He was probably much less of an anatomist than either Michael Angelo or Leonardo. His works, however, show a thorough anatomical understanding and he left many anatomic sketches.

To this group, a fourth though less brilliant artist must be added, namely, Rosso de Rossi. He has the distinction of being the first artist to attempt to prepare a volume of anatomy for artists. He did not complete the task because of his early death. His anatomical drawings were of a high order and one at least was for a long time attributed to Michael Angelo.

Toward the end of the fifteenth century we find a German physician living in Italy, editing a collection of current medical writings under the title of Fasciculus Medicinae. This book contained the first anatomic illustrations of any kind and the first wood cuts. This author was Johannes de Ketham.

On entering the third period we find Jacobi Berengario da Carpi a prominent figure. He took his Doctor's degree at Bologna and taught Surgery at Pavia, and from 1502-1527 at Bologna. He later went to Ferrara and to Rome, where he is alleged to have made much money in treating syphilis. He is credited here as being the first to use mercury in the treatment of this disease. He was a great admirer of Art and was once the possessor of Raphael's painting, "John the Baptist". He prided himself on having dissected seven hundred bodies, and was the author of two works, both fairly well illustrated.

Leprosy was rampant in Europe in the early part of the fifteenth century, and Holbein (born 1498) leaves a record of it in two of his pictures, "Lepers and the Saviour", which is in the library at Basle, and the picture of St. Elizabeth of Hungary tending the lepers at Munich. Of the latter picture Sir Bertram Windle says, that though doubt is expressed as to whether leprosy to-day is the same as that which used to be prevalent in Europe, this picture might well have been drawn from present-day patients.

This period had two great anatomists who did much to forward both anatomy and anatomical illustrating. These men were Charles Estienne and Andreas Vesalius. Vesalius was born in Brussels in 1514, and was educated in Montpellier and Paris, and later at Louvain. The chief task of his life was the revolutionizing of the teaching of human anatomy and the overthrowing of the prevailing teachings of Galen. Vesalius was the founder of modern anatomy and an active reformer of pictorial anatomic illustrations. His works were beautifully and correctly illustrated by Calcar, a pupil of the great Titian. It is said, however, that Vesalius carefully supervised the execution of these drawings. Though we now think of Vesalius in connection with a small foramen in the base of the skull, it is seldom realized that his work made such an advance over anything which had preceded them that no further improvements were made for a whole century. The works of John Banister and Helkiah Cooke, and even those of Ambrose Paré, were illustrated largely with copies of Vesalius' drawings.

Contemporary with Vesalius we find Estienne. He published a book on descriptive anatomy, but his cuts were largely vehicles for the fanciful designs of the draughtsman.

During the fourth period, that of copper engraving, many notable medical books were published. One of the most interesting works of this time was that of Godfridus Bidloo, "Anatomia Corporis". Bidloo was professor of anatomy at Hague and Leyden, and was at one time physician to William III. of England. The book made no great scientific advance. Bidloo, however, had secured the services of Gerard de Lairesse, a painter of excellent talent. The book contains two osteologic plates of interest as works of art. To quote Anderson again, "In one of these a figure standing in the entrance of a sepulchre, magnificent even in its fleshlessness, holds up an hour-glass as if to warn us that our state was separated only by the moments of passage of a few falling grains of sand; and the other, an animated skeleton retiring into the grave, not without grace and dignity, to escape a world where the stern simplicity of unclothed bones could find neither sympathy nor repose". Many of the illustrations are of a superior type.

In the seventeenth century it may be noted that two of the greatest painters of their day, Rembrandt and Rubens, left their mark in connection with medical science. Rembrandt's celebrated picture of Van Tulp demonstrating the muscles of the arm in the anatomical theatre of Amsterdam was painted in 1632. Peter Paul Rubens (1577-1640) made many accurate anatomical drawings, one of which was poorly reproduced in William Cowper's "Myotomia Reformata".

During this time there were many anatomical folios published, some containing works of Gantier and of Ladmiral, who made coloured copper-plates. The works of William Hunter and of Albinus of Leyden may also be mentioned. Cheselden produced a work which is described as a "Monument of pictorial accuracy". His drawings were largely his own work and were done with the aid of the camera obscura. John Bell's "Anatomy of the Bones and Joints", particularly in the osteologic section, was a very superior production. The plates in William Hunter's works were drawn by several very good artists. He credits the famous engraver, Robert Strange, with having been of great assistance in supervising their production.

In the last period the work produced is legion. It is difficult to say much regarding this period save to mention Max Broëdel and his school of medical illustrating at Johns Hopkins University. The medical illustrating of to-day may not be the product of the brush of any celebrated artist, but it makes up for this by our artists' comprehension of the needs of science.

BIBLIOGRAPHY

Anderson, William. An outline of the history of art in its relation to medical science. St. Thomas Hospital reports, 1886. Vol. 15 N.S.

Choulant, L. The history and bibliography of anatomical illustration. Published by the University Press, Chicago, 1920.

SOME ASPECTS OF TRACHOMA IN SASKATCHEWAN

F. S. Leeder, '28

It is doubtful if it is appreciated in Canadian medical circles just how prevalent trachoma has become. This is a question which in Saskatchewan has been under investigation by the local Department of Public Health for some years, with the collection of some rather surprising data.

Under the supervision of Dr. R. O. Davison, Director of the Division of Communicable Diseases for Saskatchewan, a detailed survey was made of an area in southern Saskatchewan, during the past summer. The area was one which was known to have in it some cases of trachoma, and which was peopled largely by immigrants from the vicinity of Odessa in South Russia, where tracoma is very prevalent. It is to be noted that the survey was of a selected group. The object of the survey was to find the total number of cases of trachoma, as well as the epidemiological history of its spread in this area. All cases found were graded according to an empirical system worked out for the purpose, and were provided with suitable medicinals and instructions for the carrying out of treatment. In certain outstanding cases the investigator gave the sufferers special individual treatments.

In all 1723 people were seen with the following findings:

Number examined	N.B. cases	+ cases	++ cases	+++ cases	%+, ++ and +++ cases
1723	25	81	80	48	12.1%

N.B.—Not definitely trachoma but suspicious.
\+ Definite first stages of trachoma.
++ Well established trachoma.
+++Old and well advanced cases.

This total of 1723 includes 14 schools with an enrolment of 250 pupils.

Number examined	N.B. cases	+ cases	++ cases	+++ cases	%+ ++ and +++ cases
250	5	18	13	5	14.4%

The increased percentage of cases in children over the percentage in children plus adults, bears out the general statements of Morax of Paris, that the child is infected by the mother, and infection passed from one adult to another is less common.

The following is a résumé of the epidemiological history of one of the families studied. It was probably the earliest case of trachoma in the area.

Mr. G—— was born in South Russia in 1854. While there, he married, and shortly after contracted "sore eyes." His parents before him had also suffered from "sore eyes." In 1898 he and his family emigrated to Gretna, Manitoba. In 1905 an area in southern Saskatchewan was surveyed and set apart as a reserve. In the spring of 1906 the family, the sons of which were now largely married, moved into this reserve, and founded the village of H——. The survey of September 11, 1925, showed 14 persons contaminated out of 37 persons living in the village, a percentage of 37.9. This shows clearly the consequences which may follow in the wake of *one* neglected case. The fact that there were found to be localized trachoma areas, with cases spreading ever outwards in ever widening circles, points to the epidemic nature of the infection.

The finding of a large number of people in a localized area, with the disease in all its various stages, provided an admirable opportunity for observing its characteristics in this country. The following is a general discussion of the condition as seen in Saskatchewan.

Dr. E. W. Fox[1] defined a trachoma as "A contagious inflammatory disease of the palpebral conjunctiva, characterized by increased thickening and vascularity, and the formation of granular elevations, or lymphoid infiltrations which undergo ulceration and subsequent cicatrization". There is some doubt as regards the necessity of granules being present. The survey of this summer showed some twenty cases which were undoubtedly trachoma, but which had instead of granules

[1]E. Webster Fox. Diseases of the eye.

the furry appearance of velvet. There seem to be then both granular and velvety types, the former being much the commoner.

Under ordinary circumstances the infection is transferred from person to person, not merely by chance contact, but by the actual rubbing of the exudate from an infected conjunctiva into a healthy one.

The disease usually has an insidious onset, and starts high up in the retrotarsal fold. The changes in the palpebral conjunctiva are slowly progressive, and tend to be accentuated along the superior tarsal margin. The area becomes thickened, roughened, and a dirty dull red; the fine blood vessels normally seen become indistinguishable in the congestion.

The importance of a thorough examination well up into the retrotarsal fold cannot be too heavily impressed. A useful instrument is the common, flat, wooden tooth pick. The lower lids can be examined by pulling them down with any of the fingers, provided that the finger is placed in the centre and as close to the margin of the lid as possible. The inspection is facilitated if the patient looks up. The following technique in the examination of the upper lid is well worth acquiring. The patient is seated on a fairly high chair in a good light. For the examination of the left eye the examiner grasps with his left hand the lashes of the upper lid near the centre, and as close to the hair roots as possible. With his right hand, in which he holds a tooth pick by means of his thumb, index, and second fingers, he pulls the lower lid down with his third and fourth fingers. This keeps the lower lid away from the upper, and with a little practice one can make it practically impossible for the patient to forceably close his eye. While exerting a pull on the upper eyelid with his left hand, the examiner quickly depresses the soft tissues above the tarsal plate with the tooth pick in his right. As the toothpick descends, the upper eyelid is pulled upwards and outwards. The eyelids should now be completely everted. The procedure is exactly similar for the right eye, except that the lower lid is held down by the third and fourth fingers of the left hand. The upper palpebral conjunctiva can be thoroughly inspected by manipulating the toothpick. It is important to examine the conjunctiva carefully, and to press out by means of the tooth pick any folds in the membrane, for it is not hard to mis take them for trachoma granules.

The importance of an early diagnosis is very great, for in its early stages it is quite amenable to treatment. Advanced conditions are very stubborn and may produce many objectional sequelae and a terminal blindness. As regards the diagnosis, the small trachoma granules are quite characteristic. They are greyish yellow bodies, varying in size, deeply seated in the conjested conjunctiva, usually over the superior tarsal margin. Slight photophobia and a sticking together of the eyelids on awakening may be complained of.

The treatment of the condition is with caustics or astringents. The following methods were adopted in the area under discussion with very gratifying results. All suspected cases were put on copper sulphate. The eye apparently bu ilds up an inurance to irritation by copper sulphate, so that incremental drop doses were used.

The affected eyes were for the first week treated with a few drops, three times a day, of a one-half of one per cent. solution of copper

sulphate in water (in practice made up by adding five drops of a ten per cent. copper sulphate solution in glycerin to one teaspoonful of water). The strength was increased every four days, until a maximum of ten to twelve drops was reached. The treatment was then carried on by the patient until cure resulted. This was found to be sufficient for the cases still in the primary stages with but few granules.

When there were many granules, a more radical form of treatment was adopted. The patient was placed on the copper sulphate as before, for a week, to accustom his eyes to the drug. The lids were then everted, and the granules pierced by a sharpened copper sulphate stick, and the eye irrigated with normal saline. Another method which gave good results was the painting of the upper eyelid, and the lower if necessary, with a ten per cent. solution of copper sulphate in glycerin, followed by fairly firm rubbing with a piece of sterile gauze on the end of a pair of forceps, and then irrigation with normal saline. This was usually done once a week, the drop treatment still being continued. Silver compounds and especially one per cent. silver nitrate, have long been in vogue for the treatment of trachoma, but to my mind the copper is superior for three reasons, the effect is at least as beneficial, there is less pain, and there are no adverse constitutional effects.

Trachoma has for years back been to most medical graduates merely a name. It is obvious from the first table, which shows 12.1 per cent. infection in a selected district, that it must now be regarded as an eye condition for which the practitioner must always be on the look out, and a condition which is going to be found quite frequently.

THICK CEREAL FEEDINGS

T. G. H. Drake, M.B.
Resident Physician, Hospital for Sick Children

Thick cereal feedings were introduced into infant feeding in 1918 by Sauer.[1] In this variety of feeding, we have a concentrated mixture of high caloric value, which on account of its tenacious character, cannot be regurgitated readily. The gastric peristalsis is able to force the thick mixture through the pylorus more readily than in the case of a liquid feeding. For these reasons the thick cereal feeding is indicated in cases of continued vomiting, such as pylorospasm, and rumination; and also in post-operative hypertrophic pyloric stenosis, in which the vomiting has not ceased entirely. On account of the high caloric value it is used occasionally in cases of decomposition.

Since the carbohydrate is largely starch, it is broken down slowly in the intestines, and hence it does not form an excessive amount of the irritating acids which produce diarrhoea. The feeding is usually the thick mixture alone, but if the infant is still on breast milk, one rounded tablespoon of the cereal feeding may be given before the nursing. The

[1]Saeur, L. W. Uses of thick farina in the treatment of pyloric stenosis, Arch. Pediatr, Vol. 35, No. 7: 385, July, 1918.

thickened mixture resulting in the stomach makes regurgitation less liable to occur.

The cereal which is usually used is farina. This is a finely granular cereal prepared from wheat, and may be procured, in bulk or in a package, from any grocery store. It contains a portion of the wheat germ, and on account of the protein content due to this, makes a more tenacious mixture than if another cereal is used.

In the preparation one quarter cup of farina is mixed with one cup of water, and cooked for 4 hours. This period of cooking is necessary to break up the cellulose envelopes surrounding the starch grains. The cooking may be carried out entirely on the stove, or during the latter part of the required time the mixture may be placed in a fireless cooker. From the above amounts about 1¼ cups of cooked farina are obtained; and this stock material may be kept for 2 days in the refrigerator.

This cooked farina is then mixed with a suitable proportion of 2% milk formula, and the whole is again cooked. 2% skim milk is used since the lowered fat content renders it more easily digestible. This partially skimmed milk is obtained by removing, with a Chapin dipper, the top 4 ounces from a quart bottle of whole milk after it has stood for 4 hours. Its caloric value is 15 calories per ounce.

In determining the amount of milk mixture necessary for the day's feedings, the following method of calculation may be used. A 2% milk and water formula of a dilution suitable to the child's age, and containing 3 ounces per pound of body weight, is first determined. For an infant weighing 7½ pounds at 1½ months of age, a mixture of 2% milk 15 ounces, water 10 ounces, would be suitable. At 3 months of age 20 ounces of 2% milk, with 10 ounces of water, could be used.

To make the thick feeding 1 heaping tablespoon (2 ounces by volume) of the cooked farina is used for every 5 ounces of the milk and water formula. Only sufficient of this final product should be made to last one day. The ingredients are mixed in a double boiler placed over the open flame, and are stirred for a few minutes until the mixture is smooth. The inner pan containing the feeding is then placed directly over the open flame, and the cooking is continued until the mixture is of the proper consistency, that is, when it will no longer run from the spoon. This requires from 30 to 40 minutes, and the mixture must be stirred continuously to prevent burning. It is then placed in a glass sealer and stored in the refrigerator, the individual feeding being removed and warmed as it is required. When properly prepared, the finished product should be smooth, non-granular, and of such a consistency that it will stand on a tongue depressor. From each heaping tablespoon of the cooked farina used, approximately 4 rounded tablespoons of the thick cereal feeding are obtained, giving 20 rounded tablespoons from the 25 ounce milk mixture when cooked, with 5 heaping tablespoons of cooked farina. Since the feeding is used for infants who are vomiting, the feeding interval is 4 hours. The infant 1½ months of age would receive $\frac{1}{6}$ of the total feeding, from 3 to 3½ rounded tablespoons every 4 hours, 6 feedings in the 24 hours. The feeding can best be given by placing a portion on a wooden tongue depressor, and forcing it well back in the mouth by means of a second tongue depressor.

The caloric value of the feeding is obtained by adding the caloric value of the milk mixture to that of the cooked farina, one heaping tablespoon of which contains 42 calories.

The feeding is increased in amount, and the percentage of milk is raised, as the infant becomes older. Sugar is added as indicated to increase the caloric value, and varied according to the character of the stools, from 1 to 1½ ounces being added to the day's feedings.

The stools are yellow or yellowish brown, pasty or slightly granular, formed or semiformed, and from 1 to 2 in 24 hours. Analysis of the stool shows that the amount of starch present is practically nil, about 99% of the carbohydrate being absorbed.[2]

Water may be given between feedings if it is not vomited. However, the infant will hardly ever become dehydrated when given nothing but the thick cereal feeding. If the patient is dehydrated or is in danger of becoming so, 10% glucose solution, 15 c.c. per pound of body weight, should be given subcutaneously 1 to 3 times in each 24 hours.

In cases of pylorospasm the efficacy of the thick cereal feeding in preventing vomiting will be increased by the administration of $\frac{1}{1000}$ of a grain of atropine in a dram of water, 10 minutes before feeding. This relieves some of the spasm of the pyloric muscle.

The infant may be weaned finally from this mixture by gradually thinning out the mixture, or by placing it directly on a milk and water formula suitable to its age.

THE DIFFERENTIAL DIAGNOSIS OF PULMONARY TUBERCULOSIS

A. W. M. White, '26

It is often stated that mistakes in differential diagnosis of pulmonary tuberculosis are made only with reference to incipient cases. It is generally considered that advanced cases are easily diagnosed. That this is not so, is clearly shown by the number of patients admitted to hospitals which treat active cases of pulmonary tuberculosis, which are later found to be non-tuberculous. At the Boston City Hospital, 11.6 per cent. of cases believed to be tuberculous on autopsy are found to be non-tuberculous. McCrae and Funk found 6% of those coming to them as diagnoised cases of pulmonary tuberculosis, did not have the disease at all. Many hospitals for consumptives have as high as 50% of their patients whose sputum is negative for tubercle bacilli. It is recognized, however, that not more than 10 per cent. of even early cases, if active, are without tubercle bacilli in their sputum, even when repeated examinations are made. With these considerations in mind, one should also remember that a false diagnosis of tuberculosis inflicts needless suffering on a patient, his relatives and friends, and entails a greater economic both to the patient and to the community.

---

[2]Mixsell, H. R. Three years experience with thick cereal feeding. Atlantic Med. Jour., Vol. 26, No. 8: 744, August, 1923.

The following are some of the conditions most often mistaken for pulmonary tuberculosis:

1. *Diseases of the Upper Respiratory Passages:*

These affections are most frequently diagnosed incorrectly as tuberculosis of the lungs. The trouble in these instances is located in the tonsils, pharynx, or accessory nasal sinuses. A patient may show a chronic cough, and occasionally expectorate muco-purulent sputum tinged with blood. During an exacerbation of the trouble, he may have some fever, lose weight, and so on. The physical examination may reveal impaired resonance at one apex. The right is frequently found so, apparently existing normally—and the patient is branded tuberculous.

In the differential diagnosis, one important principle is to be borne in mind. Active tuberculous disease is always accompanied by toxaemia. Fever and especially tachycardia are present. Patients suffering from diseases of the upper respiratory passages may have intermittent slight attacks of fever, but rarely show tachycardia. They have bad frequently occurring colds for a long time. In contrast to this, a tuberculous patient can nearly always name the exact time his cough commenced. "No patient should be pronounced sick with active phthisis unless there are found distinct signs of a tuberculous lesion, usually apical, with positive sputum—when in his case the pulse and temperature are normal, when he states that he has had frequent colds for years—and when on examination shows signs of pathological change in the nose and throat." In childhood affections of the tonsil, pharynx, and sinuses occur fairly frequently, whereas pulmonary tuberculosis is rare. Tuberculous tracheo-bronchial adenitis is relatively common as compared to phthisis, and under favourable conditions the prognosis is, as a rule, good.

2. *Collapse Induration of the Apex:*

In this condition one finds signs resembling those observed in tuberculosis, but due to respiratory obstruction resulting from adenoids and other causes. The parenchyma of the right apex usually becomes collapsed, the air is absorbed, and the lung tissue indurated, so as to resemble very closely in the physical signs produced, the apex of phthisis. These patients, however, have had an obstruction for years, the cause of which is evident on investigation. "A tuberculous patient with pronounced stigmata of the lymphatic diathesis is rare." There are lacking in these cases, also the signs of toxaemia—fever, tachycardia, languor, fatigue, loss of weight, etc.

3. *Apical Catarrh:*

One observes this especially in emphysematous patients who work at dusty trades. Well marked râles may be present at the apex, especially after vigorous coughing. Here again are lacking the signs of toxaemia. There is no apical retraction, though there may be impairment of resonance or percussion below the clavicle. At times, long observation of the patient, with oft-repeated sputum examination is necessary to differentiate the condition from one of fibroid phthisis.

4. *Acute and Chronic Pneumonic Processes:*

These are often difficult to differentiate. They are caused by various organisms, and are not usually a single pathological process. The acute types, give a history of sudden onset, with fever, cough, malaise, and pain in some region of the chest. They frequently show haemoptysis, as seen in epidemic influenzal pneumonic infection. Examination by inspection or percussion may reveal nothing abnormal, but on auscultation presents feeble breath sounds, and moist râles are heard over one lobe, usually the lower left. It is very rarely that the apex is affected, but when it is involved the diagnosis is extremely difficult. There are no tubercle bacilli in the sputum, but other organisms are found. There is often considerable muco-purulent sputum. The fever may last a month or so, and the physical signs clear up in three or four months, leaving the patient in good condition. One should remember, in differentiating these cases, that primary basal tuberculous lesions are very rare, and such patients should be considered non-tuberculous unless tubercle bacilli can be found in the sputum. Even when the apex is involved in this condition, the resonance is not lost, and X-ray findings are negative for tuberculosis.

The chronic types may have an acute or subacute onset, or may be recurrences of an acute process. The physical signs are the same as in the acute, the left lower lobe being most commonly affected. These cases are easily distinguished if one remembers that tuberculosis, besides being very rarely primary in a lower lobe, when as extensive as the signs in these cases indicate, would surely produce its characteristic signs and symptoms of toxaemia—which are lacking in these conditions. Many cases of this type are found in school age children. Contrary to the teachings of some text-books, however, which state that primary tuberculosis of the lower lobe is often found in children of this age, such a lesion should never be diagnosed tuberculous unless there are symptoms of toxaemia. Perhaps one should also demand the presence of tubercle bacilli in the sputum.

These cases do not require sanatorium treatment. The lesion may exist over a fairly long period, but as a rule, the general health suffers little, if at all.

5. *Chronic Bronchitis:*

Here the differentiation is easy if one remembers the following considerations. Excluding cases of chronic bronchitis clearly secondary to cardiac or renal disease, the patients give a history of having been subject to colds, and have been troubled with chronic cough and expectoration for years, with little impairment of the general health. A tuberculous patient can practically always give a definite date of the commencement of his cough, fever, night sweats, anoresia, loss of weight, etc.—which symptoms are not concomitants of a chronic bronchitic process. One should hesitate in diagnosing tuberculosis in a patient who has coughed for years, and whose general health is good—unless some evidence of toxaemia exists with tubercle bacilli present with sputum.

In chronic bronchitis, the harsh bronchovesicular, or bronchial breath sounds are audible nearly all over the chest. Râles are present, par-

ticularly at the bases, and bilaterally. In phthisis, it is the apices alone which give physical signs, unless the disease is very advanced. When it is so, the patient presents a very different picture from that observed in chronic bronchitis, and then tubercle bacilli are almost sure to be found in the sputum.

6. *Bronchiectasis:*

In this condition, the patient presents more closely the picture of advanced tuberculosis and the diagnosis is much more difficult. He has a long history of cough and expectoration, dating perhaps from a pneumonia, a pleurisy, or following a general anaesthetic. The cough is periodic, with longer or shorter remissions when it is slight; but large quantities of sputum are expectorated without much effort. Cough and expectorations are often influenced by posture. There may be a slight fever, 99°.5 in the afternoon, but no subnormal morning temperature is present, as in phthisis. Unlike pulmonary tuberculosis the pulse is normal except in long standing or advanced cases when cardiac dilatation produces tachycardia, dyspnoea, cyanosis, etc. Patients with bronchiectasis may have haemoptysis, and there may occur fairly severe haemorrhages. There is also present a polymorphonuclear leucocytosis which is uncommon in phthisis.

The following are some of the main features in differentiating between bronchiectasis and pulmonary tuberculosis. (*a*) With such profuse expectoration tubercle bacilli almost surely will be found on sputum examination, if the case be tuberculous. (*b*) Patients who are tuberculous and have such a degree of expectoration are febrile and cachectic, and are very likely to have laryngeal or intestinal complications. (*c*) The points on physical examination are important: (i) The lesions in bronchiectasis are most marked in the middle or lower lobes. The percussion note at the bases is influenced by the amount of secretion in the bronchi—being dull previous to, and resonant following expectoration. (ii) When bronchiectasis is present in an upper lobe, which is unusual—there is usually dullness below the clavicle, on percussion, but resonance above it—the reverse of phthisis. (iii) Bronchiectasis is often unilateral; but patients with large tuberculous cavities in one lung, practically always show affection of the opposite side. (iv) Bronchiectatic cavities remain the same size for years; but tuberculous cavitation associated with signs of active phthisis, tend to increase decidedly in size. (v) In multiple bronchiectasis there is practically normal lung tissue between the cavities. In tuberculosis the process is continuous with the apical disease. (vi) The X-ray picture of bronchiectasis shows remarkably little as compared with the physical findings. In tuberculosis the picture shows more extensive findings than the physical signs would lead one to expect.

7. *Pleural Vomicae:*

This condition consists in localized collections of pus in the pleural cavity, and oftenest in the interlobar fissures. They may originate in the thorax or abdomen, but finally burrow and burst through into a bronchus. Pleural vomicae may follow pneumonia, empyema, surgical operation, or labour. There is always a history of an acute process with

finally the sudden expectoration of large quantities of purulent material. The amount of sputum decreases in a few days, and the condition becomes chronic. The temperature drops, but remains subfebrile, going up and down inversely as the amount of material expectorated. The patient may become emaciated. He may show haemoptysis—and at this stage phthisis is suspected. On physical examination one is inclined to diagnose fluid in the pleural cavity. Paracentesis fails to confirm this. The apex of the lung is usually unaffected—a fact of great importance. There is flatness on percussion over the affected area, and the breath sounds may be feeble or cavernous, depending on the amount of purulent material in the cavity. There are often numerous consonating râles. One excludes pulmonary tuberculosis by the history of an acute process, the clear apex, and the absence of tubercle bacilli in the sputum.

8. *Lung Abscess:*

The history in this condition is helpful, as lung abscess often follows an aspiration pneumonia which may follow any operation on the upper respiratory passages, such as tonsillectomy. It may result after the occurrence of a pulmonary embolism of a septic nature. The fever is hectic and there is sweating, emaciation and spasmodic cough. There is present in this instance a large amount of sputum of a brownish colour, in which haematoidin crystals and elastic tissue may be found. Haemoptysis often occurs. In all cases, the clear apex, the character of the sputum with absence of tubercle bacilli, are the differential diagnostic points.

9. *Gangrene of the Lung:*

Where this process follows aspiration pneumonia, embolism or entry of foreign bodies into the bronchi, the diagnosis is not difficult. Preceded by bronchiectatic symptoms, with perhaps haemoptysis, the resemblance to phthisis is more approximated. In gangrene of the lung the patient shows a septic type of temperature, prostration, and characteristic sputum which separates into three layers, lung tissue being found in the lowest. It is very uncommon that the sputum of phthisis is foetid unless there is a complicating gangrene; but this combination is very rare.

10. *Malignant Pulmonary Neoplasia:*

Carcinoma and sarcoma of the lung, bronchi, pleura or glands, sometimes resembles tuberculosis of the lungs considerably. There is often cough, mild fever, loss of weight, and perhaps haemoptysis. Malignant disease of the lung never pursues a very rapid course. Every symptom and sign should be considered in differentiating their condition. There is often pressure on the superior vena cava, causing enlargement of the veins of the chest, shoulders and lateral aspects of the neck. There may be differences in the radial pulse on either side. While many symptoms simulate phthisis, there is rarely tachycardia in early malignancy. There is often dyspnoea with tumor, but this is rare in early phthisis. Loss of weight occurs earlier in phthisis than in pulmonary neoplasia. Later, when cachexia is marked, the phthisical

patient shows a hectic flush, whereas the other shows a waxy yellow colour. Percussion yields a flat note over a new growth, with perhaps, resonance above it. A clear resonant apex should always cause one to hesitate before making a diagnosis of pulmonary tuberculosis. When the flatness on percussion is at the base, tuberculosis should not have the first consideration. A pleural effusion of tuberculous origin may, however, be simulated, but it should be easily excluded by history course and physical signs. Auscultation is useful. Feeble breath sounds accompanied or associated with no râles, together with a very flat percussion note, should make one very suspicious of tumor. The feebleness of breath sounds is due in this case to compression of a bronchus by the tumor, with resulting collapse of alveoli. These signs in the absence of tachycardia, fairly well exclude phthisis, even when fever, cough, etc., and perhaps haemoptysis are present.

One should further note the position of the trachea and heart. In phthisis with extensive involvement of the lung, these structures are pulled to the affected side, with tumor they are pushed to the opposite side. When malignancy is present there is pleural effusion in 50% of cases. It may be serous, sero-sanguineous or even purulent. This increases the diagnostic difficulties. Bloody fluid is often present in phthisis, though less often on first paracentesis than it is in malignancy. Sometimes fragments of the tumor can be found in the centrifuged specimen. Many eosinophiles in the differential smear are a suspicious sign of tumor. With carcinoma the effusion usually extends over the lower half of the side of the chest involved. In inflammatory effusions there is resonance above the fluid and in hydro- or pyopneumothorax there is shifting dullness. Replacement of fluid by air, or induction of a pneumothorax makes possible much more conclusive findings in X-ray examination, in differentiation of neoplasia from phthisis.

11. *Actinomycosis:*

Early in actinomycosis of the lung there is cough, loss of weight, slight fever and so on, as in phthisis. The sputum is negative until the process bursts into a bronchus, when the fungus may be found. When the pleura is reached by the actinomycotic process, symptoms of pleurisy with effusion may arise, and the patient is treated for this, or for empyema. In advanced pulmonary actinomycosis, the constitutional symptoms are very much like phthisis. In early or latent cases one should remember that actinomycosis usually begins in a middle or lower lobe. When it is on the upper lobe, the differential diagnosis is impossible, because the sputum is negative, as it may be in early phthisis. In a few weeks, however, a fluctuating swelling may appear in the chest wall, and the fungus may be demonstrated in pus removed from this—or perhaps in the sputum if a bronchial ramification has been communicated with.

12. *Pulmonary Conditions in Cardiac Patients:*

Organic heart disease, especially when associated with a mitral stenosis often causes such symptoms as cough, expectoration (and the latter may be blood tinged), mild fever and loss of weight. The amount of blood in the sputum may vary from small to fairly large amounts,

though the latter is rare. It is due in these cases to the increased pressure in the pulmonary capillaries caused by obstruction to the blood flow through the mitral orifice. In cases of myocardial failure, haemoptysis may be present due to infection or embolism, usually the former. In embolism there is sudden onset of acute pain in the chest; dyspnoea, cynosis and haemorrhage. Infection is as a rule due to thrombosis and the symptoms are less acute.

One should remember that occasionally patients with mitral stenosis may be hoarse, and have a cough—and this is to be explained by pressure on the left recurrent laryngeal nerve, by the dilated left auricle. In chronic cases associated with mitral stenosis, showing signs and symptoms resembling tuberculosis, one should remember that dyspnoea is much more pronounced in cardiac than in phthisical patients. Cardiac dyspnoea is often relieved by a few days rest, cardiac cough is increased by cold weather, and does not respond to sedative drugs, e.g., codeine, as does the tuberculous. In phthisis, the heart is not enlarged as a rule, or if it is, it is very late in the disease..

On physical examination one may find areas of collapsed lung, catarrh or pulmonary oedema. The apices are clear as a rule, though occasionally they are dull, due to the pathological change referred to as brown induration. Râles are usually confined to the bases. A cardiac murmur is often a very important differential point. The snapping first sound at the apex, the systolic shock on palpation, and the outline of the heart, in cases where a mitral stenosis exists, is useful in diagnosis. Auricular fibrillation may be present. It is important to remember that mitral stenosis and pulmonary tuberculosis seldom co-exist. It is a wise rule never to diagnose phthisis in a patient showing disease of the mitral valve with cardiac hypertrophy, unless tubercle bacilli are found in the sputum. This is also a good rule to follow in any patient showing signs and symptoms resembling those seen in phthisis, but which can be equally well explained on a basis of cardiac disease.

13. *Syphilis of the Lungs:*

This disease is extremely rare and is very difficult to diagnose clinically. The symptoms resemble those of phthisis; cough, slight fever, expectoration and loss of weight occurring. It is, however, often found, in cases reported in literature, in the lower or middle lobe, with the apex clear. Other signs of syphilis should be looked for very carefully. The Wasserman reaction may assist, but one should also remember that the two diseases often co-exist, and when they do, the sputum is apt to be scanty—making examination for tubercle bacilli more difficult. The therapeutic test may be useful, but antisyphilitic treatment is stated to be harmful in tuberculosis.

14. *Hyperthyroidism:*

The syndrôme in this disease has often been mistaken for that of phthisis. Tachycardia, frequent sweating, loss of weight, slight irregular fever, tendency to fatigue, menstrual disturbance—symptoms occurring in hyperthyroidism, are also suggestive of pulmonary tuberculosis—especially if they are associated with a more or less chronic cough. Severe cases of Graves' disease with its cardinal signs and symptoms, are

not likely to be mistaken for phthisis, unless the conditions co-exist. Patient showing tachycardia, sweating on the least exertion or excitement, and more in the daytime, slight tremor, dermographia, etc., should never be diagnosed as tuberculous, unless there is found a definite pulmonary lesion. There may be collapse induration co-existing with hyperthyroidism, and in this case prolonged observation, with many sputum examinations may be necessary.

Symptoms of hyperthyroidism are frequently present in phthisis, but they have been stated to indicate a good prognosis as a rule. Within certain limits hyperactivity of the thyroid gland is incompatible with progression in phthisis. Thus there is no reason for a hasty diagnosis, for even if the two diseases do co-exist, the outlook is good in the majority of cases. For the same reason, neurocirculatory asthenia, which is considered to be due to hyperthyroidism, is not likely to develop into tuberculosis.

BIBLIOGRAPHY

Fishberg—Pulmonary Tuberculosis.
Jour. Am. Med. Assoc.—1908, 4, 205; 1915, 64, 11; 1917, 25, 645; 1919, 72, 161; 1921, 76, 581.
Am. Jour. Med. Science—1913, 146, 313.
Am. Review of Tuberculosis—1917, 1, 1; 1917, 1, 289.
Br. Med. Journal—1920, 1, 591.
Archiv. Med.—1919.

FACULTY NOTES

Medical At Home

The date of the Medical At Home has been officially set for February 2nd. Due to the efforts of the Medical Society, the dance is to be held at the King Edward Hotel, in the Crystal Ballroom. The At Home committee are sparing no effort to make the dance highly successful. The following have consented to act as Patrons and Patronesses: Dean and Mrs. Primrose, Dr. and Mrs. Hendry; Dr. and Mrs. John Oille, and Dr. and Mrs. C. L. Starr.

Graduation Dance

The 2T6 Graduation Dance was held at the King Edward Hotel, on Friday, December 18th, and was very well attended. It was patronized by Dean and Mrs. Primrose, and Dr. and Mrs. E. S. Ryerson. Dancing was conducted in the Pompeian Room, the music being furnished by Herb. Smith's orchestra. There were many interesting novelties on the programme, and the supper in the Pickwick Room left nothing to be desired. Much credit is to be given to the very efficient committee, consisting of Messrs. Campbell, Vivian and Murray, who supervised the evening.

Canadian Medical Association

At the last annual meeting of the Canadian Medical Association the By-Laws were amended, to provide a reduced membership fee for

recent graduates, namely, $3.00 for the first year from date of graduation to December 31 of the following year; $5.00 for the second year, and the full fee ($10.00) thereafter. This membership fee covers all privileges, including the Association Journal.

Deceased

Members of '26 deeply regret the death of W. G. Bright which occurred early in December at his late residence in Wiarton, Ont. A popular member of the class, he was oblidged to withdraw during the fourth year through illness.

The occurrence of the Christmas holidays has decreased the number of Faculty Notes in this issue, and more are promised for the February JOURNAL.